TO STUDY THE CLINICAL PROFILE AND OUTCOME OF PATIENTS WITH SEPSIS AND LEFT VENTRICULAR SYSTOLIC DYSFUNCTION

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DECLARATION BY THE CANDIDATE

I hereby declare that the thesis entitled **"TO STUDY THE CLINICAL PROFILE AND OUTCOME OF PATIENTS WITH SEPSIS AND LEFT VENTRICULAR SYSTOLIC DYSFUNCTION"** submitted towards partial fulfillment of the requirements for the award of **MD IN GENERAL MEDICINE** degree to BFUHS is a compilation of original work carried out by me under the supervision of **Dr. Vipin Kumar,** Associate Professor, Department of Medicine, **Dr. Dinesh Jain**, Professor, Department of Medicine & **Dr. Bhupinder Singh,** Former Associate Professor, Department of Cardiology. No part of this thesis has formed the basis for the award of any degree previously.

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Dedicated

to

my Family

&

Teachers

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LIST OF ABBREVIATIONS

BA	BRONCHIAL ASTHMA
CKD	CHRONIC KIDNEY DISEASE
CAD	CORONARY ARTERY DISEASE
CLD	CHRONIC LIVER DISEASE
CHF	CONGESTIVE HEART FAILURE
CRP	C- REACTIVE PROTIEN
CNS	CENTRAL NERVOUS SYSTEM
COPD	CHRONIC OBSTRUCTIVE PULMONARY DISEASE
CVA	CEREBROVASCULAR ACCIDENT
CABG	CORONARY ARTERY BYPASS SURGERY
DM	DIABETES MELLITUS
DBP	DIASTOLIC BLOOD PRESSURE
ESR	ERYTHROCYTE SEDIMENTATION RATE
ET SECRETION	ENDOTRACHEAL SECRETION
GCS	GLASGOW COMA SCALE
GIT	GASTRO INTESTINAL TRACT
ICU	INTENSIVE CARE UNIT
LVSD	LEFT VENTRICULAR SYSTOLIC DYSFUNCTION
LVDD	LEFT VENTRICULAR DIASTOLIC DYSFUNCTION
LVEF	LEFT VENTRICULAR EJECTION FRACTION
PCT	PROCALCITONIN
qSOFA	QUICK SOFA SCORE
RA	RHEUMATOID ARTHRITIS
RTI	RESPIRATORY TRACT INFECTION
RSA	ROAD SIDE ACCIDENT
SOB	SHORTNESS OF BREATH
SBP	SYSTOLIC BLOOD PRESSURE
SIRS	SYSTEM INFLAMMATORY RESPONSE SYNDROME

SOFA	SEQUENTIAL (SEPSIS- RELATED) ORGAN FAILURE ASSESSMENT SCORE
TURP	TRANSURETHRAL RESECTION OF THE PROSTATE
TURBT	TRANSURETHRAL RESECTION OF BLADDER TUMOR
UTI	URINARY TRACT INFECTION

ABSTRACT

Introduction: Sepsis is defined as life threatening organ dysfunction caused by a dysregulated host response to an infection and its a major cause of death worldwide. Presence of Myocardial Dysfunction in Sepsis makes management of sepsis challenging because of the complex hemodynamic changes occurring in these patients. Early detection and optimized treatment increases the likelihood of survival for patients with sepsis and cardiac dysfunction. We aim to study the association of Left ventricular Systolic dysfunction with the clinical outcome of sepsis patients.

Aim of Study: 1) To study the clinical profile of patients with sepsis and left ventricular systolic dysfunction (LVEF <50%)**.** 2) To study the outcome of patients with sepsis and left ventricular systolic dysfunction (LVEF<50%)

Materials & methods: A prospective, single center, observational study was carried out in Medical ICUs/HDU at Dayanand Medical College and Hospital, Ludhiana of patients with Sepsis (Definition & criteria as per Third International Consensus Definitions For Sepsis and Septic shock,2016 –Sepsis 3 guidelines) and Left Ventricular Systolic Dysfunction LVEF<50%. Duration of the study was 1 year from March 1, 2021 to March 1, 2022. All patients were included in the study after complying with inclusion and exclusion criteria and written informed consent for admission into study was taken from the patient or legal representative of the patient. The details of patients demographic data, clinical history/ examination, laboratory investigations, 2d-echocardiography were noted onto the performa and later on analyzed for the conclusions of the study.

Results: Total 130 cases of sepsis with Left ventricular systolic dysfunction with LVEF<50% were taken for the study. Male gender, old age, decreased urine output, altered sensorium as presenting complaints, co-morbid conditions like chronic kidney disease, diabetes mellitus are associated with mortality outcome. Respiratory tract and urinary tract are major sources of sepsis. Low Glasgow coma score at presentation, High SOFA score at presentation and decreased LVEF significantly associated with sepsis and mortality associated with it.

Conclusion - With increasing age, higher SOFA score at presentation, with lower LVEF% mortality outcome worsens in patients. Having comorbid conditions like Diabetes, Hypertension, Chronic kidney disease, coronary artery disease increases the risk of worse outcome in patients with sepsis and cardiac dysfunction.

Keywords: Sepsis, Septic Shock, Left Ventricular Systolic Dysfunction, Left Ventricular Ejection Fraction (LVEF%)

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INTRODUCTION

Sepsis Definition

Sepsis has been recognized in one form or another since 1000 BC, first described by the Islamic philosopher Ibn Sina (also known as Avicenna) as a blood and tissue decay accompanied by fever[1]. It has been further described by Boerhave, von Liebig, Semmelweis, Paster, Lister, Lennharz and most recently Roger Bone. The definition of sepsis has changed over the years since Roger Bone and colleagues at the American College of Thoracic Physicians and Conference in Critical Care Medicine (SCCM-ACCP) in 1991 [2]. Previously, sepsis was defined as SIRS due to infection. “Severe” sepsis was associated with organ dysfunction, hypoperfusion, or hypotension. "Septic shock" is associated with arterial hypotension despite adequate fluid therapy.

1991 criteria for sepsis, severe sepsis and septic shock

The following definitions derived from the 1991 Consensus Conference of the American College of Chest Physicians and Society of Critical Care Medicine. Infection is defined as the presence of microorganisms or tissue invasion by microorganisms.

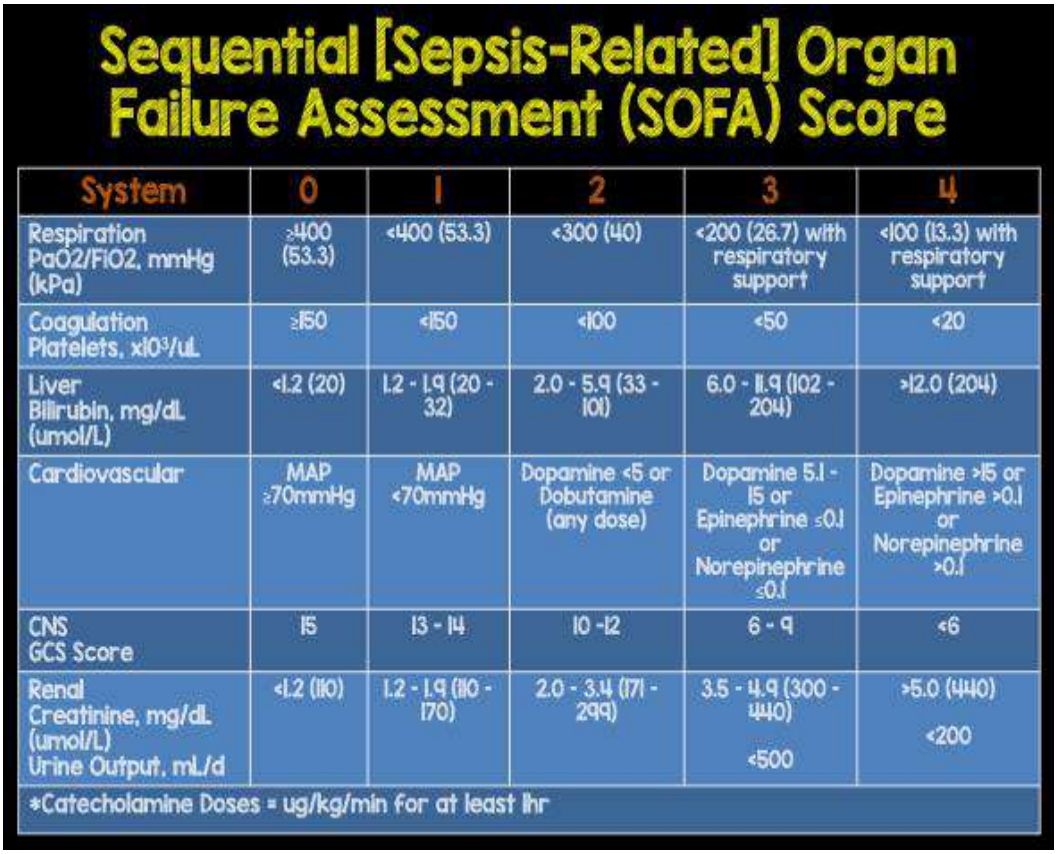
- Sepsis -The systemic inflammatory response (SIRS) to infection, manifested by at least two of:
    - Temperature of >38°C or <36C
    - Heart Rate of >90 beats per minute
    - Respiratory rate of >20 breaths per minute or partial pressure of CO2 of <32mmhg
    - WBC of > 12,000 per ml or <4000 per ml, or > 10% immature (band) forms
- Severe sepsis -Severe sepsis is defined as sepsis associated with organ dysfunction, hypotension or hyperfusion. Hypoperfusion abnormalities of end organs may include lactataemia, oliguria or an alteration in mental status.

- Septic shock -Septic shock is defined as sepsis associated with hypotension and perfusion abnormalities despite the provision of adequate fluid (volume) resuscitation. Perfusion abnormalities include lactic acidosis, oliguria or an acute alteration in mental status. Patients with septic shock who are receiving inotropic or vasopressor therapy might still exhibit perfusion abnormalities, despite the lack of hypotension.

The definitions of sepsis and septic shock were updated in January 2016 to identify patients at increased risk of adverse outcomes, particularly requiring intensive care unit (ICU) treatment or at high risk of death [2,3]. Sepsis-3 defines sepsis as “life-threatening organ dysfunction resulting from an uncontrolled host response to infection” [4]. Organ dysfunction is indicated by an increase in the SOFA score of 2 points or more, which is associated with hospital mortality of 10% or more.

Importantly, Septic Shock, a subset of sepsis, is clinically identified by a vasopressor requirement to maintain a mean arterial pressure of 65 mmHg or greater and serum lactate level greater than 2 mmol/l in the absence of hypovolemia [5]. Septic shock has hospital mortality of >40%.

The current definition recognizes the severity and potential lethality initiated by invasion by pathogens, resulting in a process in which the body's defense response has its own detrimental effects [6]. These pathophysiological responses can lead to complex organ failure, aptly described as the “Four Horsemen of the Septic Apocalypse”, usually due to a combination of cardiovascular, cellular, coagulation and endothelial dysfunction [6]. It is recognized that the mortality rate increases as treatment is delayed, so timely diagnosis is essential to start treatment early and obtain the best outcome [7,8].



Picture 1: Parameters of Sequential Organ Failure Assessment Score (SOFA) [Adapted from sepsis-3 guidelines][2]

The recent working group on sepsis definitions suggested a new screening tool, the quick SOFA score. The quick SOFA assigns one point for each of hypotension (systolic blood pressure≤100 mmHg), tachypnoea (respiratory rate of≥22 per minute) and altered mental status (Glasgow Coma Scale score of 13 or less) and is suggested as a simple way to identify patients with suspected infection who are likely to have a prolonged stay in intensive care or to die in hospital. When any two of these variables are present simultaneously, the patient is considered qSOFA positive. Numerous studies have investigated the potential use of the qSOFA as a screening tool for sepsis.The results have been contradictory as to its usefulness. Studies have shown that qSOFA is more specific but less sensitive than having two of the four SIRS criteria for early detection of

infection-induced organ dysfunction [9-12]. Neither SIRS nor qSOFA are ideal screening tools for sepsis.

Epidemiology of Sepsis

Comprehensive epidemiological data on the global burden of sepsis are not available despite the high associated mortality. Preliminary extrapolation of data from high-income countries suggests that there are 31.5 million sepsis cases and 19.4 million severe sepsis cases worldwide each year, potentially 5.3 million people die each year [13]. Knowledge of morbidity and mortality from sepsis in low- and middle-income countries is only an estimate because of the lack of data and the difficulty of obtaining estimates at the population level in those regions [14–16].

According to recently published analysis, ie. Global Burden of Disease, Injury and Risk Factors Study (GBD 2017), there were 48.9 million sepsis cases and 11 million sepsis-related deaths worldwide in 2017, accounting for approximately 20% of all deaths worldwide [17]. Roughly 1.7 million adults in the U.S. develop sepsis each year and approx 270,000 die each year [18]. Importantly, sepsis remains the leading cause of intensive care unit (ICU) admissions in low- and middle-income countries (LMICs). Mortality rates in this region reach 80% [19]. There have been an estimated 11.3 million cases of sepsis in India alone, with 2.9 million deaths (297.7 per 100,000 population in 2017). In 2016, India ranked only 145 out of 195 countries in the Global Burden of Disease Healthcare Access and Quality Index [20]. The World Health Organization (WHO) recently recognized sepsis as a global health priority [21]. Sepsis and septic shock are serious health problems that affect millions of people worldwide each year, killing between 1 in 3 and 1 in 6 of those affected [22,23].

Sepsis can be caused by a variety of microorganisms. Pneumonia, urinary tract infections and bloodstream infections were common foci of infection in India [24] as well as in studies conducted in other developed countries [25]. The most common source of sepsis was the respiratory tract (67.4%) followed by the abdomen (21.8%) .Positive isolates were retrieved

in 69.6% (n = 2069) of patients with sepsis; two thirds of these patients had Gram-negative microorganisms isolated and half had Gram-positive microorganisms.[26]

Pathophysiology of sepsis

There is a general acceptance that the pathophysiology of sepsis is considered as an initial hyper inflammatory phase that lasts for several days, followed by a more protracted immunosuppressive phase [27,28]. These two phases are associated with increased mortality and the current death distribution indicates two peaks; one of which manifests during the early phase albeit to a lesser extent, and another second peak after 2–3 months that continues to increase over the next 3 years [29,30]. The peak mortality rate in the early period is attributed to an overwhelming inflammatory response, also known as a “cytokine storm,” which comprises fever, refractory shock, inadequate resuscitation, and cardiac or pulmonary failure. Mortality during the latter period is due to protracted immunosuppression with secondary infections, culminating in organ injury and/or failure. Despite sophisticated ICU care resulting in improved mortality, patients may still die at a later period or after several years owing to the persistent immunosuppression, immune dysfunction, or chronic catabolism [27].

The initial hyperinflammatory phase of the disease is a series of steps leading to a cytokine storm. The pathogenesis of sepsis involves early activation of both innate and adaptive immune responses [5]. Detection of an invading pathogen initiates an inflammatory response (IR). Immune host cells express pattern recognition receptors (PRPs) on the extracellular surface and in the cytoplasm. Toll-like receptors (TLRs) and Nod-like receptors (NODs). TLRs are essential for pathogen detection, recognizing ‘pathogen-associated molecular patterns’ (PAMPS) from pathogens, and recognizing ‘damage-associated molecular patterns’ (DAMPS) from damaged endogenous cells. The overstimulation of TLRs by DAMPS may promote the IR in sepsis release of proinflammatory cytokines like TNFa,

IL-1,IL-6 etc. NODs detect pathogens that invade the cytosol, leading to the formation of inflammasomes, promoting the production of inflammatory cytokines IL-1b, IL-18, and caspases which are programmed in cell death[7]. This promotes an inflammatory state with activation of leukocytes, complement and coagulation pathways that underpin the endothelial, cellular and cardiovascular dysfunction that characterizes sepsis [31]. Others indicate that disruptions in the normal homeostatic mechanisms of the immune and neuroendocrine systems during sepsis, alter cellular energy processes, disrupting endothelial and epithelial functions, which can ultimately cause dysfunction at the organ level [32]. In sepsis, inflammatory and hemostatic pathways intersect, stimulating both inflammatory and coagulation cascades. This cascade ranges from mild thrombocytopenia to fulminant DIC.

Cardiac Dysfunction In Sepsis

Organ dysfunction in sepsis results from reduced oxygen delivery and use by cells as a result of hypoperfusion. Hypoperfusion is caused by cardiovascular dysfunction. The incidence of septic cardiomyopathy varies from 18 to 60% in different studies. The pathophysiology of sepsis-induced cardiac dysfunction can be summarized as myocardial circulatory disorders, direct myocardial dysfunction, and mitochondrial dysfunction. Impaired blood flow to the myocardium is associated with microvascular dysfunction, endothelial damage, and ventricular-arterial separation. The mechanism of direct myocardial inhibition consists of inhibition of β-adrenergic receptors and some myocardial inhibitors (eg cytokines and nitric oxide) [33].

Host immune system recognizes infection through recognition of pathogen-associated molecular patterns (PAMPs), such as lipopolysaccharide (LPS), lipoteichoic acid, flagellin and DNA in bacteria, mannan in fungi, and single- or double-stranded RNA in viruses. These mediators bind to pattern-recognition receptors (PRRs), such as toll-like receptors (TLRs) that are expressed on the surface of host cells. These PRRs are essential for initiating host immune defenses against invading pathogens and mediating

PAMP recognition. They also serve as receptors for endogenous danger signals by identifying various damage-associated molecular patterns (DAMPs) as potent activators of the innate immune system [34-36].

The proinflammatory response induced by infection is normally balanced by anti-inflammatory cytokines. However, the normally effective inflammatory response to infection becomes systemically dysregulated during sepsis due to significantly imbalanced cytokine responses referred to as a cytokine storm. TLRs and PAMPs activate intracellular signal-transduction pathways that lead to the nuclear translocation of nuclear factor-κB (NF-κB) and the increased transcription of inflammatory mediators[28]. Among these, proinflammatory cytokines such as tumor necrosis factor-alpha (TNF-α) and interleukin-1-beta (IL-1β), chemokines, and lipid mediators play major roles in the inflammatory process[37]. The production of excess antimicrobial products and inflammatory mediators elicits the generation of reactive oxygen and nitrogen species, superoxide anion (O_2^-), and nitric oxide (NO), causing adjacent tissue damage and an amplified inflammatory reaction[38,39]. The DAMPs released during tissue damage include heat-shock proteins, high-mobility group box 1 (HMGB1), histones, and oxidized lipoproteins. Other cytosolic constituents such as adenosine triphosphate (ATP) and mitochondrial products, including mitochondrial DNA (mtDNA), can also contribute to the activation of innate immunity that initiates SIRS and a sepsis-like state. Excessive production of DAMPs can activate inflammation, create a vicious circle, and finally facilitate cardiac dysfunction, multiple organ failure (MOF), and death.

The most important feature of septic cardiomyopathy is that it is acute in onset and reversible. Second , the low LVEF is accompanied by normal or low LV filling pressures(unlike in cardiogenic shock) with increased left ventricular compliance[40].Multiple studies have shown both systolic and diastolic dysfunction with decreased stroke volumes and increased end- diastolic , end-systolic volumes in sepsis[41,42].Because of arterial and venous dilation (induced by inflammatory mediators) and

consequent reduced venous return , a state of hypotension and distributive shock is produced by sepsis. Above said changes along with microvascular thrombosis can result in hypo-perfusion of tissues and organs.

There are significant changes in the endothelium with a violation of barrier function, vasodilation, increased leukocyte adhesion and creation of a procoagulant state. This leads to the accumulation of edematous fluid in the interstitial space, body cavity and subcutaneous tissue. In the lungs, protein-rich fluid accumulates in the interstitial spaces and alveoli, disrupting the alveolar-endothelial barrier. This can lead to ventilation and perfusion mismatch, hypoxia, decreased pulmonary compliance, and acute respiratory distress syndrome (ARDS). In the kidney, reduced renal perfusion and acute tubular necrosis combine to result in acute renal injury. In the gastrointestinal tract, increased mucosal permeability leads to bacterial translocation across the intestinal wall and intestinal autophagy by luminal enzymes. Inhibition of bilirubin clearance in the liver results in cholestasis. Changes in thinking indicate dysfunction in the central nervous system. Endothelial changes weaken the blood-brain barrier, resulting in an influx of toxins, inflammatory cells, and cytokines. Subsequent changes in the form of cerebral edema, neurotransmitter dysfunction, oxidative stress and white matter damage result in the clinical spectrum of septic encephalopathy. Sepsis is known to cause a catabolic state. Muscle is rapidly and significantly broken down to produce amino acids for gluconeogenesis, which nourishes immune cells. [43]

The initial pro-inflammatory state of sepsis often leads to a long-term immunosuppressive state. As a result of apoptosis, the number of T cells (helper and cytotoxic) is reduced and the response to inflammatory cytokines is reduced. Post-mortem studies of ICU patients who died of sepsis demonstrated a global depletion of CD4+ and CD8+ T cells, particularly in lymphoid organs such as the spleen [6,28]. Therefore, the immune system of a person with sepsis cannot provide an effective immune response to secondary bacterial, viral, or fungal infections.

Diagnosis of sepsis and challenges

Sepsis differs from the localized microbial infection in terms of host response which is dysregulated generalized, with signs and symptoms, which are nonspecific [44]. For example, fever is a typical clinical reaction indicating the onset of a host response. At the same time, hyperthermia is common in critically ill patients, which is not always indicative of the presence of infection. Moreover, tachycardia and leukopenia are present in critically ill patients or may be due to other underlying pathological conditions. Most often, sepsis can sometimes be underdiagnosed in patients with hypoxia, and low platelet count but lacking evidence of infection or overdiagnosis can occur in postoperative patients on antibiotic therapy presenting with fever [44,45]. In this regard, the criteria for SIRS (Systemic Inflammatory Response Syndrome) appear to be unreliable, so its use in the diagnosis of sepsis is not recommended [46].

Early diagnosis of sepsis and timely therapeutic intervention are critical to improving clinical outcomes and reducing mortality [47]. Traditionally, serological analysis and molecular methods have been used to diagnose sepsis. Diagnosis of sepsis can be difficult because it is further complicated by non-specific signs and symptoms and there is no standard test to confirm the diagnosis [45].

In addition to symptom-based diagnosis, culture reports from body fluids, preferably blood, is the most confirmatory and reliable means of diagnosis, but the assay time of 24 h to 48 h restricts their early clinical usefulness [48]. Unfortunately, around 30–40% of presumed sepsis cases of infected patients are found to be culture-negative, which may be attributed to prior/ongoing antibiotic therapy or colony-forming units below detectable levels [46]. Hence, due to the aforementioned diagnostic challenges, tracking using valid biomarkers which will allow for early therapeutic intervention, and improved patient outcomes is a key focus area in sepsis diagnosis.

Biomarkers for sepsis are helpful in the identification of presence or absence of infection, the severity of disease condition, and the patient's

response to treatment [44]. The biomarkers in sepsis management are often evaluated for diagnostic, prognostic, monitoring, surrogate and stratification purposes [49]. A small set of biomarkers have been successful in clinical diagnosis for sepsis, which includes CRP, PCT, and Interleukin-6 (IL-6) [50,51].

C-reactive protein (CRP) is a widely studied acute-phase protein, produced only by hepatocytes in response to inflammation or tissue injury triggered and has high sensitivity, lack of specificity, and restricts its application in sepsis diagnosis. CRP is released either in response to infection or cytokine stimulation, in particular IL-6, during inflammation. The exact role of CRP in inflammation is unknown, but it may bind with bacterial components, enabling clearance by macrophages [50]. The level of CRP in a healthy individual is less than 10 mg/L, but the initial rise is reported in 4–6 h after tissue injury and peaks by several hundred folds within 24–48 hr [29]. CRP exhibits good correlation with the severity of the infection and thus helps in early diagnosis in sepsis patients [52].

Procalcitonin (PCT) is probably a more specific marker than CRP, which is secreted during systemic inflammation caused by bacterial infection [53]. PCT is a prohormone (peptide precursor) of calcitonin secreted by the thyroid gland of healthy individuals, and the presence of bacterial toxins can significantly increase serum levels of PCT. In a meta-analysis of 30 studies (3,244 patients), procalcitonin showed a sensitivity of 77% and a specificity of 79% for sepsis in critically ill patients [54].

Apart from PCT, tumor necrosis factor-α (TNF-α), a potent cytokine biomarker, is also involved in a wide range of pathological and physiological processes, acting as an endogenous mediator for inflammation and immunity.

Proven complexities and variable times of individual biomarker expression makes a single biomarker for sepsis diagnosis inappropriate, especially in critically ill patients.

Management of sepsis and challenges

Guidelines for sepsis management are focused on three major components (1) haemodynamic stabilization, (2) infection control, and (3) modulation of the septic responses [46]. Other interventions include non specific measures of organ support, such as oxygen therapy, mechanical ventilation, hemodynamic support, corticosteroids, and renal replacement therapy [55]. Sepsis management requires multimodal therapeutic approaches and is based on severity. A mild form with single organ system dysfunction can be managed by moderate support, while multiple organ dysfunction requires invasive therapies [56]. Haemodynamic management of sepsis related hypotension should commence immediately. The 2018 update of the Surviving Sepsis Campaign guidelines introduced the 'Hour-1 Bundle' which recommends treatment with intravenous fluids, measurement of serum lactate concentration(elevated levels >2.5mmol/L) as a marker of illness severity, administration of vasopressors, obtaining blood cultures and administering broad-spectrum antibiotics, all within the first hour[57].

Although broad-spectrum antibiotics are integral in the management of sepsis, a major challenge associated with antibiotic therapy in sepsis is resistance by pathogens that adversely affects sepsis outcomes and increases mortality rates by approximately two-fold [58]. Early recognition and treatment, particularly early administration of effective antibiotics, are associated with decreased mortality[59,60]. The original influential data came from Kumar's group in Canada and have recently been confirmed by study of the New York State Department of Health Database. In the New York study, risk-adjusted in-hospital mortality was 28% in patients in whom antibiotic treatment was delayed 12hr compared with 23% in patients who received antibiotics within 1hr, representing a 22% increase in relative risk of death. Taken together, these studies suggest that the relative risk of death from sepsis or septic shock may increase by between 4 and 8% for each hour of delay in administering effective antibiotics[59,60]. Delays in

empiric treatment for sepsis and bacteremia increase mortality as well as length of stay and cost [61,62].

Fluid administration is a cornerstone in the management of hemodynamic instability, optimizing its administration is still challenging[63]. To avoid delayed hemodynamic stabilization, the early initiation of a vasopressor is recommended, but most of the vasopressors are lacking information on safe and effective doses [64]. Sepsis-induced hypotension results from different pathological processes that require different treatments. Hypovolemia from intravascular volume loss due to increased vascular permeability requires fluid resuscitation, vasodilatation affecting both the venous capacitance vessels and the arterial resistance vessels is treated with vasopressor agents, and sepsis-related myocardial dysfunction may require specific inotropic agents[62]. Not all commentators agree with the Surviving Sepsis Campaign Guideline recommendation to commence intravenous fluid resuscitation of sepsis-induced hypo-perfusion at a rate of 30 mL/kg over 3 hr [65]and currently the bundle is under review because of concerns that its uncritical application may be harmful[54].In response to the Early Goal Directed Therapy (EGDT) study, a more restrictive approach to fluid therapy is currently being evaluated in a randomized controlled trial (RCT) [66].

There is an established biological rationale for the administration of adjunctive corticosteroids in the management of patients with septic shock. Corticosteroids act through two mechanisms: immune modulation and cardiovascular modulation. Studies demonstrated that treatment with lower doses of corticosteroids, typically hydrocortisone in a dose of 200 mg per 24 h, results in more rapid reversal of shock[67-70].

Although antibiotic therapy is central in sepsis management, inappropriate therapy within the first 24 h leads to eight-fold higher, in-hospital mortality. In contrast, a 74% higher progression in the inflammatory response, in inadequate empirical antibiotic therapy compared to adequate, has been reported previously[71]. Other challenges associated with antibiotic

therapy are hemodynamic alterations, leading to sub therapeutic dosing, and variations in resistance patterns amongst different regions [65]. Despite the use of supportive therapy and timely administration, antibiotics are often ineffective and show little impact on lowering the mortality rate due to sepsis [72].

The immune paralysis associated with sepsis predisposes critically ill patients to secondary infections, including breakthrough infections, by multidrug-resistant (MDR) bacteria. Therefore, these patients require specific strategies to restore immune response function along with antibiotic treatment and standard supportive care. Therefore, these adjuvant therapies may help the immune system by preventing immune paralysis or attenuating the inflammatory response [56,72]. Emerging data on adjuvant therapies ranging from in vitro blood purification techniques to a variety of pharmacological approaches including pro-inflammatory cytokine suppression, immunomodulatory and antioxidant activities represent potentially active preclinical models and emerging studies in sepsis.

Apart from antimicrobial agents, novel adjunctive therapies such as synthetic antimicrobial peptides, anti-inflammatory agents, immune-modulators, blood purification and antioxidants are being explored and have shown some additive benefits on survival in sepsis patients [73]. Many of these alternative interventions for sepsis fail to reproduce efficacy in clinical practice. This disparity can be explained by sepsis-induced pharmacokinetic alteration, leading to poor bio-distribution with compromised efficacy and safety of commonly used therapeutic agents, thus increasing the risk of treatment failure or resistance development [74,75]. The incidence of sepsis is increasing especially in elderly population with more comorbidities. It is now estimated that sepsis is a leading cause of mortality and critical illness worldwide. The epidemiological data regarding sepsis, septic shock and organ involvement is mainly from western literature. Data from India, especially Northern India is less when compared to western data. In order to improve management and survival of sepsis

patients, robust data on regional epidemiology and critical clinical variables are essential in addition to early identification of focus of infection. Acknowledging the need to support evidence based clinical decisions and subsequent protocols, this study is being conducted in tertiary care hospital to study the clinical profile and outcome of patients with sepsis and left ventricular systolic dysfunction.

AIMS AND OBJECTIVES

1) To study the clinical profile of patients with Sepsis and LV Systolic Dysfunction (LVEF<50%).
2) To study the outcome of patients with Sepsis and LV Systolic Dysfunction (LVEF<50%).

REVIEW OF LITERATURE

In a study done by Daniel R Ouellette and Sadia Z Shah (2005-2010) on comparison of outcomes from sepsis between patients with and without pre-existing left ventricular dysfunction: a case-control analysis. In this study, 197 patients with pre-existing left ventricular systolic dysfunction and sepsis were compared to 197 case-matched patients with normal prior cardiac function and sepsis. In-hospital mortality rates ($P = 0.117$) and intubation rates at 24 hours ($P = 0.687$) were not significantly different between cases and controls. There was no correlation between the amount of intravenous fluid administered over the first 24 hours and the PaO_2/FiO_2 ratio at 24 hours in either cases or controls. Mortality risk factors for cases included intubation status, compliance with a sepsis bundle, a source of infection other than the lung, and the initial mixed venous oxygen saturation.[76]

In a study done by S.Chatterjee et. al. (2006-2011) on epidemiology of adult population sepsis in India out of 4711 admissions, 282 cases had severe sepsis. ICU mortality, hospital mortality and 28 day mortality were 56%, 63.6%, 62.8% respectively. Predominant infection site was respiratory tract. Most common organisms were gram negative microbes. The most common organism was Acinetobacter baumannii. Median apache-2 score on admission was 22 (interquartile 16-28) and median length of ICU stay was 8 days. Severe sepsis attributable mortality was 85%.[77]

In a study done by M. Mukhyaprana Prabhu et.al (2010-2012), on prognosis of left ventricular systolic dysfunction in septic shock patients. Out of 66 patients with septic shock, the source of infection is not known in 42.8% of patients. Among the known sources, the most common source of infection was pneumonia (24.2%). 24.2% patients reported left ventricular systolic dysfunction (LVSD) on the first day of admission. Out of 66 patients of septic shock, mortality rate was 48.48% and among them survivors had better LVEF than non survivors signifying that the low ef is a measure of mortality in these patients. Non-survivors exhibited lower mean blood pressure, lower ejection fraction. Among the inotropes used for maintaining

blood pressure, 44.1% of the survivors were resuscitation with norepinephrine and combination of noradrenaline and dopamine (59.4%) being the most often used in non-surviving septic shock patients.[78]

In 2012 Juan N Pulido conducted a study on clinical spectrum, frequency, and significance of myocardial dysfunction in severe sepsis and septic shock and found that the frequency of myocardial dysfunction in patients with severe sepsis or septic shock was 64% (n=68). Left ventricular diastolic dysfunction (LVDD) was present in 39 patients (37%), LV systolic dysfunction in 29 (27%), and right ventricular dysfunction in 33 (31%). There was significant overlap. The 30-day and 1-year mortality rates were 36% and 57%, respectively. There was no difference in mortality between patients with normal myocardial function and those with left, right, or any ventricular dysfunction.[79]

TT Paary et al (2013-2014) conducted a study on clinical profile and outcome of patients with severe sepsis treated in ICU in India. Out of 1162 patients, 356 patients had severe sepsis. Incidence of severe sepsis was 30.6% and mortality rate was 51.6%. APACHEII (23.37 ± 9.47) and SOFA (7.58 ± 4.05) scores at admission were high. Most common source of infection was respiratory tract (37.2%) followed by urinary tract (10.3%) and intra-abdominal (9.5%) infections. About 63% of patients required ventilator support, 25.5% of patients required vasopressor support despite adequate fluid resuscitation and one third of patients required renal replacement therapy (35.7%). Hematocrit, total leukocyte count, serum bilirubin and sofa scores were significantly higher among non-survivors.[80]

In a study done by Jalal HKT et.al. (2017) on epidemiology of sepsis in multi-disciplinary ICU in south India, out of 497 patients, 59.8% patients were male; the majority were above 60 yr (range 18 to 92 yr). 76.3% of patients had comorbid disease. Hypertension was the most common comorbid condition (62%) followed by Diabetes mellitus (51.3%), chronic kidney disease was found in 132 patients (26.6%) and Coronary artery disease in 121 patients (24.3%). 186 patients (46.5%) had single organ

involvement, 140 (35%) patients had 2 organ involvement, 74(18.5%) patients had more than 2 organ involvement. Septic shock was found in 155 patients (31.2%). Renal involvement was the most common organ involvement found in 279 patients (59.9%). Most common source for sepsis was the respiratory system found in 230 patients (46.3%), followed by urinary tract infection in 117 patients (23.5%). The study shows a significant association between SOFA score and mortality (p-value 0.001) 52% of mortality happened in group of patients with SOFA score more than 15 and mortality was 28 % in group with a SOFA score of 10-15.[81]

In a study done by Kim S, Lee JD et. al (2015-2018), on association between LV systolic dysfunction and mortality in patients with septic shock, total of 208 patients were enrolled in the study. Pneumonia was the leading cause of septic shock (60% patients), followed by urinary tract infection (13.5% patients). In-hospital mortality was 49.5% in total. APACHEII score of the non-survivors was higher than that of the survivors, the CK-MB and troponin-t were higher in the non-survivors than in survivors. Risk of in-hospital mortality of patients with severe LV systolic dysfunction with LVEF<30% was significantly higher than that of patients with normal Left ventricular systolic function, although the risk of in-hospital mortality did not change in patients with mild or moderate systolic dysfunction[82].

In a study done by Abou Dagher et. Al. (2018) on outcome of patients with systolic heart failure presenting with sepsis, out of total 174 sepsis patients, 87 had previous history of Congestive Heart Failure (CHF). 43.7% of cases had respiratory infection, 33.3% had urinary tract infection,11.5% had skin infection, 4.6% had GI tract infection. Patients with CHF were more likely to be admitted to ICU (59.8% vs 48.8%) as compared to the control group. Patients with CHF had higher in-hospital mortality (57.5% vs 34.5%) and a higher 72 hour mortality (10.3% vs 4.6%).[83]

In a study conducted by Saraschandra Vallabhajosyula et al. (2018) new-onset heart failure and mortality in hospital survivors of sepsis-related left ventricular dysfunction. During this 8-year period, 434 patients with 206

(48%) patients having LV dysfunction were included. The two groups had similar baseline characteristics, but those with Left ventricular dysfunction had worse function as demonstrated by worse LV ejection fraction, cardiac index, and LVDD. In the 331 hospital survivors, new-onset acute decompensated heart failure hospitalization did not differ between the two cohorts (15% vs. 11%). The primary composite outcome was comparable at two-year follow-up between the groups with and without LV dysfunction (p=0.24). Persistent LV dysfunction was noted in 28% hospital survivors.[84]

In a study conducted by Meghan M. Cirulisa et al (2018) right-to-left ventricular end diastolic diameter ratio in severe sepsis and septic shock this is a retrospective chart review of patients with severe sepsis and septic shock admitted to a medical intensive care unit (ICU) at a single tertiary care hospital. Patients were identified by icd-9 codes: 995.92 for severe sepsis and 785.52 for septic shock; and had to have an echocardiogram within 48 hr of ICU admission. Increased RV/LV was defined as RV/LV ≥ 0.9. Left and right-sided chamber dimensions were measured according to American society of echocardiography guidelines. They included 146 consecutive ICU patients admitted with septic shock (72) or severe sepsis (74). There was no significant difference in ICU mortality in patients with RV/LV≥ 0.9 versus RV/LV < 0.9 (p = .49).[85]

In a study done by Andrew M. N. Walker et al (2018) on prevalence and predictors of sepsis death in patients with chronic heart failure and reduced LVEF results. They conducted a prospective cohort study of 1802 patients with CHF and left ventricular ejection fraction ≤45% attending CHF clinics in 4 United Kingdom hospitals between 2006 and 2014. Mode of death was defined over a 10.3-year follow-up period (mean 4 years). Competing risk regression defined mode-specific hazard ratios for sepsis, other non cardiovascular, progressive heart failure, and sudden cardiac death in relation to established heart failure prognostic markers. Of 737, 173 (23.5%) were due to sepsis; respiratory tract infections accounted for 69.9% (n=121) of these events. Those who died from sepsis were older, had higher

platelet counts, and had a higher prevalence of chronic obstructive pulmonary disease than those who died from other causes. Sepsis death was independently associated with older age, greater prevalence of chronic obstructive pulmonary disease, male sex, lower log serum vitamin D, and higher platelet count than non sepsis death. Established heart failure prognostic markers exhibited different patterns of association with sepsis death, other non cardiovascular death, progressive heart failure death, and sudden cardiac death.[86]

Study done by C.M. Raibhoge and Kiran Mali on clinical profile of sepsis in patients admitted in ICU (2018-2019), out of 64 cases, majority of the cases were from 31-40 years and 51-60 years (21.9%). Mean age of the study population was 51.56 ± 19.48 years. Males were predominant (57.8%) with male to female ratio as 1.378:1. Sputum culture was found positive in 24(37.5%) cases followed by urine culture in 17(26.6%), pus culture in 6(9.4%) and CSF as well as biopsy culture found positive in 2 cases each i.e. 3.1%. Streptococcus pneumoniae was predominantly found in microorganisms (31.3%) followed by E.Coli (28.1%), Staphylococcus aureus (9.4%), and Klebsiella pneumoniae (6.3%). Most commonly involved site of infection was lungs (43.8%) followed by urinary bladder (15.6%), kidney (10.9%) and brain (9.4%) cases. Death rate was 42.2% in our study. Mean duration of ICU stay was 5.84±3.16 days. Majority of the patients stayed in ICU for less than 7 days i.e. 39(60.9%) and remaining 25 i.e. 39.1% stayed in ICU for more than 7 days.[87]

In a retrospective study done by Dong Geum Shin et al. (2020) On factors associated with abnormal LVEF in patients with sepsis in ICU, 366 sepsis patients were included. Sepsis patients were classified into 3 groups according to LVEF% in echocardiographic assessment done within 48 hours of diagnosis of sepsis. LVEF<50% (n=36), LVEF 50-70% (n=252), LVEF>70% (n=78). Decreased LVEF occurred in 36(10%) patients and hyperdynamic LVEF>70% occurred in 78 (21%) patients. Of 366 patients, 103 (28%) patients died. Mortality rates were similar in three groups,

however mortality rates were significantly higher in patients with abnormal ejection fraction (decreased or increased vs normal). Length of hospitalization was significantly shorter in patients with hyperdynamic ejection fraction. Bnp and initial troponins were highest in patients with reduced ejection fraction.[88]

In 2021 Vincent X Liu et al. conducted a study on the profile of infection and sepsis patients hospitalized through the emergency department. Using multivariable logistic regression, the association of each sign and symptom with four outcomes was evaluated: sepsis versus suspected infection diagnosis, hospital mortality, ICU admission, and time of first antibiotics (> 3 vs ≤ 3hr from presentation). A total of 10,825 signs and symptoms were identified in 6,148,348 clinical documentation fragments. The most common symptoms overall were as follows: dyspnea (35.2%), weakness (27.2%), altered mental status (24.3%), pain (23.9%), cough (19.7%), edema (17.8%), nausea (16.9%), hypertension (15.6%), fever (13.9%), and chest pain (12.1%). Fever, present in only a minority of patients, was associated with improved mortality For common symptoms, the peak time of symptom onset before sepsis was 2 days, except for altered mental status, which peaked at 1 day prior to presentation.[89]

In study done by N.E.Hammond et.al (2022), on estimate of sepsis prevalence and outcome in adult patient in ICU ,a total of 35 ICUs (85.7% private, 14.3% public) participated, enrolling 680 patients (median age, 60 years [interquartile range, 24 years]; 62.1% men). The most common primary diagnosis was renal disease, and common comorbidities were diabetes (44.0%) and chronic renal failure (11.6%). The sepsis prevalence on the study day was 382 of 677 patients (56.4%). Prevalence by sepsis-3 and sepsis-2 definitions was 33.2% and 46.2%, respectively, with minimal concordance. In patients with sepsis, the most common microorganisms were bacterial (77.9%) followed by fungal (14.1%) infections. Approximately 45% of infections were caused by multidrug-resistant organisms. Mortality at 30 days after the study day and rates of discharge against medical advice

were significantly higher in the sepsis cohort compared with the cohort without sepsis, respectively.[90]

In a study done by Fabian Muehlberg et al (2022) on Characterization of critically ill patients with septic shock and sepsis-associated cardiomyopathy using cardiovascular MRI, fifteen patients with septic shock were prospectively recruited from the intensive care unit. Individuals received a cardiac MR scan (1.5 T) within 48 h after initial catecholamine peak and a transthoracic echocardiography at 48 and 96 h after cardiac MR. Patients were given 0.2 mmol/kg of gadoteridol for extracellular volume quantification and late gadolinium enhancement imaging. Laboratory analysis included high-sensitive troponin T and N terminal pro brain natriuretic peptide levels. Nine patients had impaired systolic function with left ventricular ejection fraction (LVEF) < 50%.and three individuals had preserved LVEF (66.9 ± 6.7%). Global longitudinal strain was impaired in both subgroups (LVEF impaired: 11.0 ± 1.8%; LVEF preserved: 16.0 ± 5.8%; P = 0.1). All patients with initially preserved LVEF died during hospital stay; in-hospital mortality with initially impaired LVEF was 11%. Upon echocardiographic follow-up, LVEF improved in all previously impaired patients at 48 and 96 h. Patients with impaired systolic function had increased T2 times as compared with patients with preserved LVEF. Left ventricular GLS was decreased in all study individuals with impaired LVEF and less impaired with preserved. T1 mapping showed increased T1 times in patients with LVEF impairment as compared with patients with preserved LVEF.Extracellular volume values were elevated in patients with LVEF impairment as compared with patients with preserved LVEF.[91]

In a study done by Sidharath Duggar et al. (2023) Left Ventricular Systolic Dysfunction Associated With Increased Mortality Among Patients With Sepsis and SepticShock? ,a retrospective cohort study comprising all adult patients admitted to the medical ICU from January 1, 2011, through December 31, 2020, with sepsis and septic shock as defined by the Third

International Consensus Definitions for Sepsis and Septic Shock guidelines. All adult patients with sepsis or septic shock who underwent transthoracic echocardiography within 3 days from admission to the medical ICU were included. We divided patients into five groups based on LV ejection fraction (LVEF). In addition to univariate analysis, we also performed multivariate logistic regression analysis adjusting for patients' baseline characteristics and severity of illness. The primary outcome was the association between each classification of LVEF and in-hospital mortality.[92]

MATERIALS AND METHODS

A prospective, single center, observational study to be carried out at Dayanand Medical College and Hospital, Ludhiana of patients with Sepsis (Definition & criteria as per Third International Consensus Definitions For Sepsis and Septic Shock,2016 –Sepsis 3) with Left Ventricular Systolic Dysfunction. Duration of the study will be from March 1 ,2021 to March 1, 2022 .Only patients admitted in Medical ICU/HDU will be included in the study. Sepsis patients with left ventricular systolic dysfunction with LVEF <50% (evaluated using 2d-echocardiography) will be included in the study. These patients will be categorised into 3 groups- patients with LVEF<30%, LVEF30-40% and LVEF41-50%. All patients will be included in the study after complying with inclusion and exclusion criteria and written informed consent for admission into study has been taken from the patient or legal representative of the patient.

SOURCE OF DATA

Patients' demographic data, present and past history, clinical/ laboratory data echocardiography report, treatment and hospital course will be observed prospectively and will be noted on a proforma and observed/analysed for the study.

METHOD OF COLLECTION OF DATA

- **Inclusion Criterion:**

  -Age equal to or more than 18

  -Patients admitted in Medical ICU/HDU

  -Patients with suspected/ documented infection.

  -Criteria of diagnosing sepsis in patients with suspected/ documented infection according to sepsis-3 guidelines 2016.

  -Sepsis patients with Left ventricular systolic dysfunction with LVEF<50% evaluated using 2d-echocardiography (if clinicaly indicated and prescribed by primary physician) will be included in the study.

-With written consent from patient/legally authorised representative to be a part of study

- **Exclusion Criterion:**

  -age <18 years

  -Pregnant patients

  -poor echo window

  -patients with LV EF>50%

  -patients with congenital heart disease

  -Patients with significant valvular heart disease (more than mild valvular disease)

Duration of Study/ Number of Cases: Duration of study will be from March 1,2021 to March 1, 2022

Outcome: Primary outcome of the study to be observed in terms of:

- Discharge/Mortality outcome
- Total hospital stay
- 30-day mortality outcome

Statistical Analysis:

Data will be described in terms of range,mean+standard deviation (+SD),median, frequencies(number of cases) and relative frequencies(percentages) as appropriate. All statistical calculations will be done using (statistical package for the social science) SPSS 21 version (SPSS incc.,Chicago,IL,USA) statistical program for Microsoft Windows.

OBSERVATIONS AND RESULTS

Table 1: Distribution of Age groups.

Age Group (in years)	No. of cases	Percentage
< 30	13	10.0%
31-40	9	6.9%
41-50	14	10.8%
51-60	31	23.8%
61-70	38	29.2%
> 70	25	19.2%
Total	130	100.0%

Minimum age in this study was 18 and Maximum age was 100 with mean 57.28 yrs with SD of 16.69. Majority 38/130 (29.2%) patients belonged to the age group of 61-70yr.

Figure 1: Distribution of Age groups.

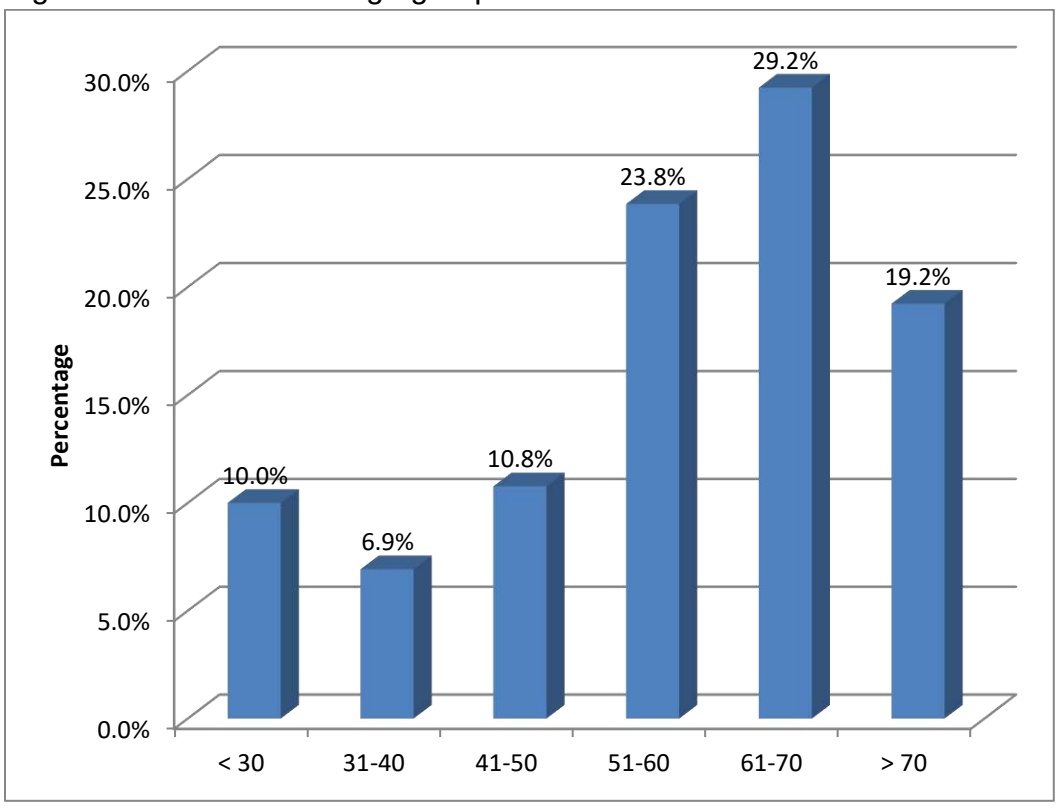


Table 2: Gender Distribution

SEX	No. of cases	Percentage
F	45	34.6%
M	85	65.4%
Total	130	100.0%

45/130 cases (34.6%) were females and 85/130 (65.4%) were males with male to female ratio 1.88:1

Figure 2: Gender Distribution

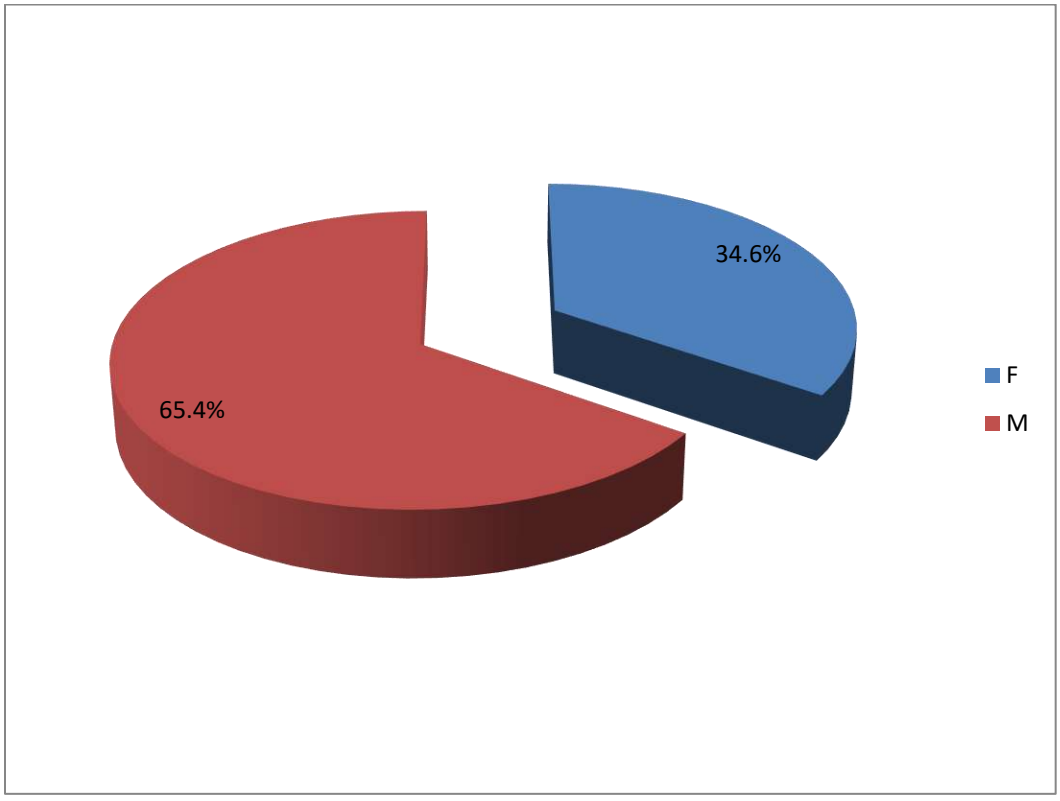


Table 3: Presenting Symptoms

Symptom	No. of cases	Percentage
Fever	67	51.5%
Cough/Sputum	38	29.2%
Pain Abdomen	15	11.5%
Decreased Urine Output	34	26.2%
Burning/ Increased Frequency Of Urination	13	10.0%
Loose Stools	5	3.8%
Altered Sensorium/ Seizure	48	36.9%
Shortness of Breath (SOB)	61	46.9%
Nausea/Vomiting	11	8.5%

67/130 (51.5%) cases had Fever as presenting symptom. Shortness of breath was second most common presenting symptom in 61/130 (46.9%) cases followed by altered sensorium/seizure in 48/130 (36.9%) cases, then cough/sputum production in 39/130 (29.2%) cases, 34/130 (26.2%) cases had decreased urine output.

Figure 3: Presenting Symptoms

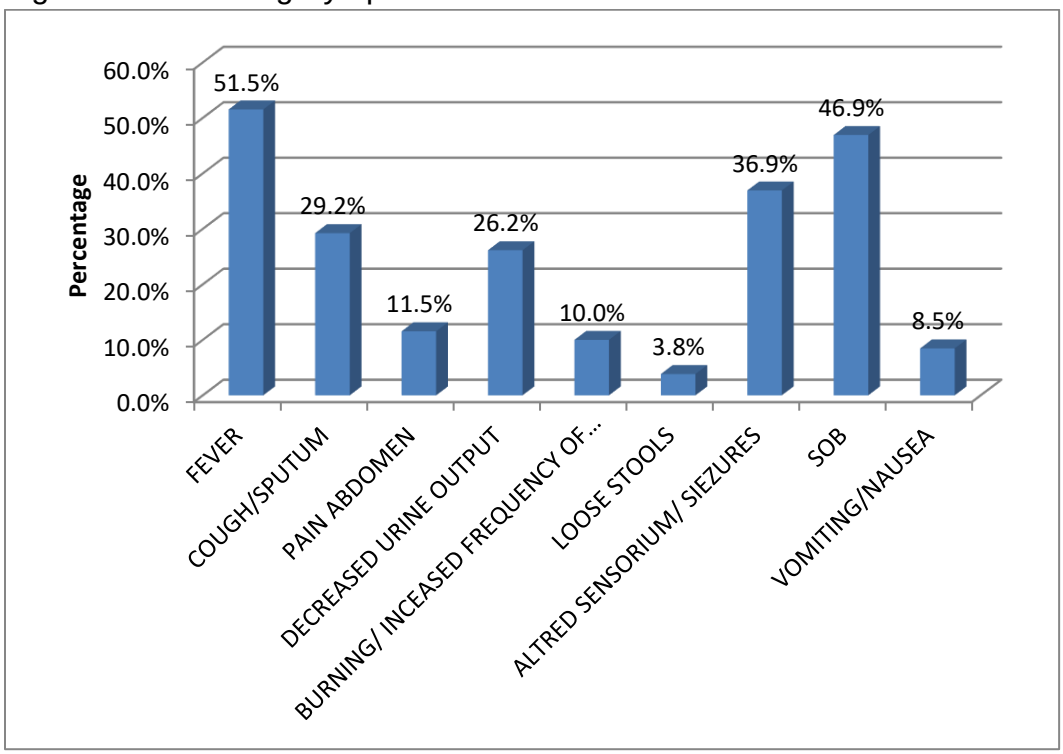


Table 4: Other presenting symptoms

Other Presenting Complaints	No. of cases	Percentage
Abdomen Distension	2	1.5%
Black Stools	1	0.8%
Blood In Sputum	2	1.5%
Blood In Urine	1	0.8%
Blood In Vomitus	1	0.8%
Body Swelling	1	0.8%
Celphos Poison	1	0.8%
Chest pain	6	4.6%
Constipation	1	0.8%
Dizziness	1	0.8%
Domestic Fall	1	0.8%
Facial Swelling	1	0.8%
Foot Gangrene	1	0.8%
Headache	2	1.5%
Jaundice	1	0.8%
Leg Swelling	1	0.8%
Lethargy	7	5.4%
Loss Of Appetite	4	3.1%
Non Healing Wound	1	0.8%
OPC Poisoning	1	0.8%
Pedal Swelling	5	3.8%
Per Vaginum Bleed	1	0.8%
Periorbital Swelling	2	1.5%
Right Hemiparesis	3	2.3%
Skin Tightening	1	0.8%
Slurred Speech	1	0.8%
Slurred Speech, Dizziness	1	0.8%
Snake Bite	1	0.8%
N	78	60.0%
Total	130	100.0%

Patient presented with other associated symptoms like chest pain in 6/130 (4.6%), lethargy 7/130 (5.4%), pedal swelling 5/130 (3.8%), loss of appetite in 4/130 (3.1%) ,right hemiparesis 3/130 (2.3%) cases, abdominal distension 2/130 (1.5%), blood in sputum 2/130 (1.5%), black colored stools, blood in urine, blood in vomitus, body swelling, celphos poisoning, constipation, dizziness, foot gangrene, jaundice, leg swelling, non healing wound, OPC poisoning, snake bite 1/130 (0.8%) each.

Figure 4: Other presenting symptoms

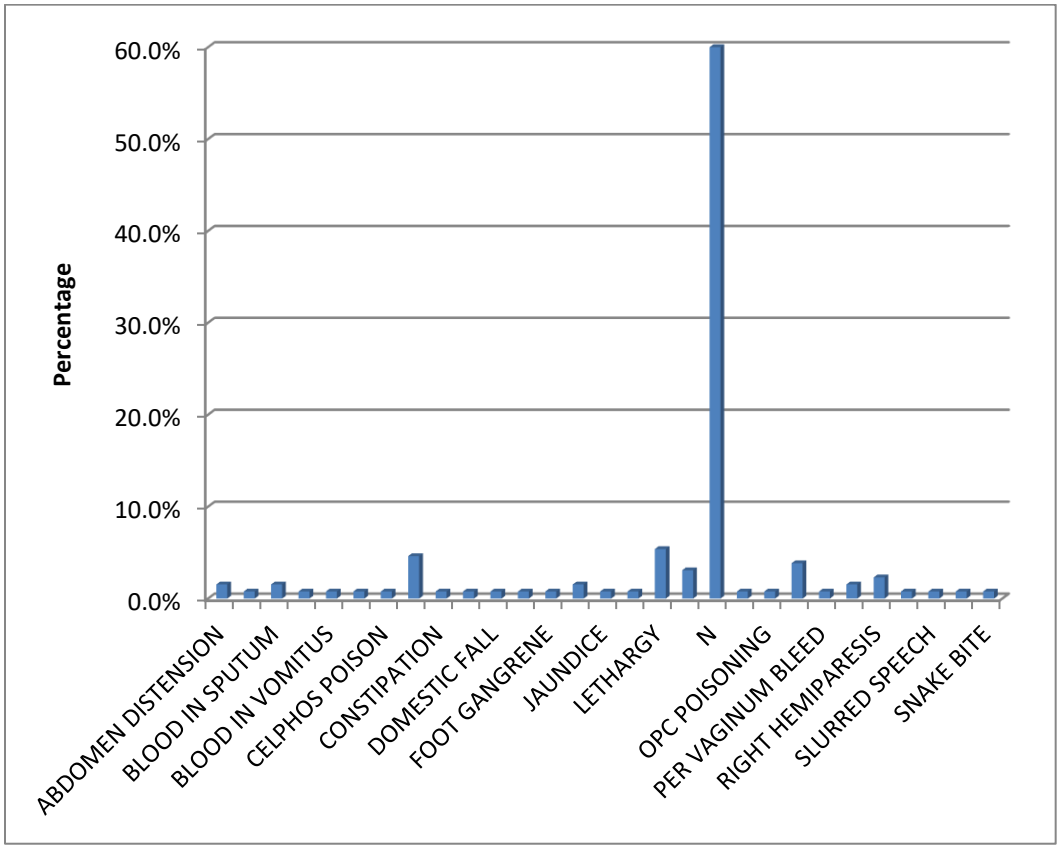


Table 5: Comorbidities

Comorbidities	No. of cases	Percentage
DM	74	56.9%
HTN	69	53.1%
CLD	6	4.6%
CKD	20	15.4%
COPD/BA/Any Resp Disorder	12	9.2%
CAD	35	26.9%
Hypothyroidism	15	11.5%
CVA	8	6.2%

Maximum number of cases had Diabetes mellitus 74/130 (56.9%) cases , Hypertension 69/130 (53.1%) cases , CAD 35/130 (26.9%) cases, and CKD 20/130 (15.4%) cases. 15/130(11.5%) cases had Hypothyroidism, 12/130 (9.2%) cases had Chronic Resp. illness.

Figure 5: Comorbidities

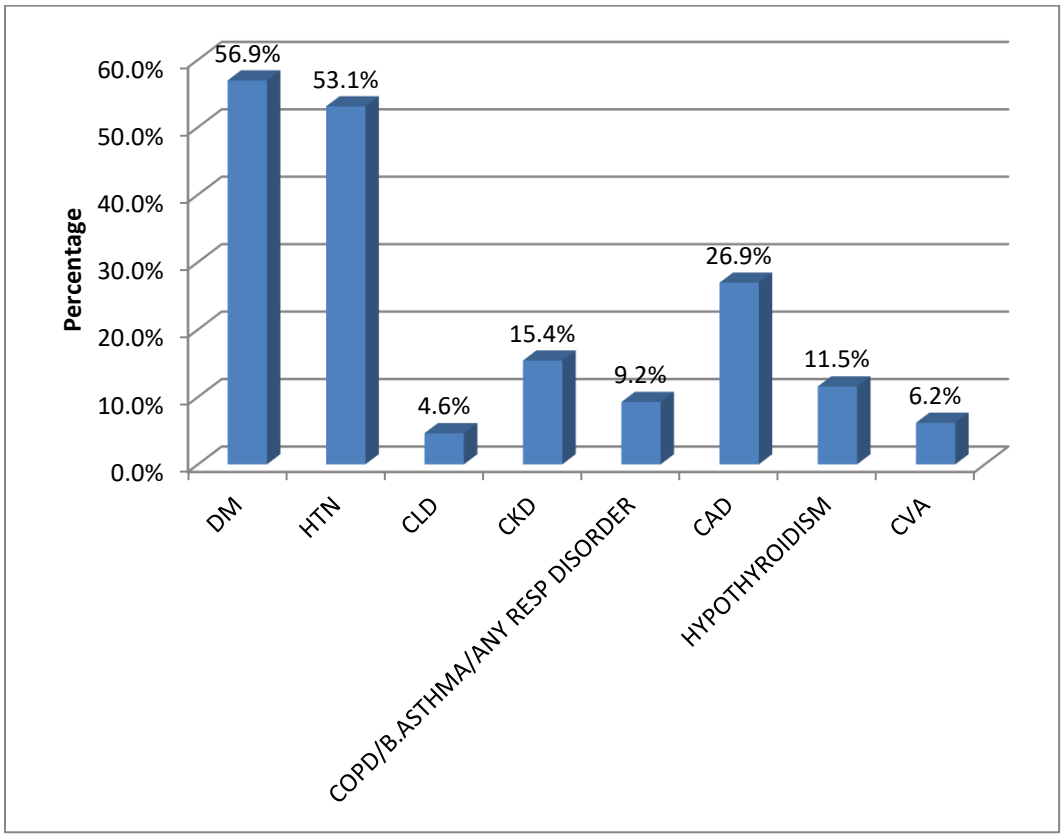


Table 6: Other past Medical history

OTHER MEDICAL HISTORY	No. of cases	Percentage
B-CELL LYMPHOMA	1	0.8%
BED RIDDEN	1	0.8%
BIPOLAR DISORDER	1	0.8%
BULLOUS PEMPHIGOID	1	0.8%
BURNS, HERNIA, BED RIDDEN	1	0.8%
CA PROSTATE	1	0.8%
DEMENTIA	1	0.8%
DEPRESSIVE ILLNESS	3	2.3%
EXTRA PULM. TB	2	1.5%
HCV REACTIVE	1	0.8%
HYPERTHYROIDISM	1	0.8%
INTELLECTUAL DISABILITY	1	0.8%
KYPHOSCOLIOSIS	1	0.8%
MOTOR NEURON DISEASE	1	0.8%
OLD PULM TB.	2	1.5%
P-ANCA VASCULITIS, RA	1	0.8%
PARKINSON	2	1.5%
PLHIV	2	1.5%
POST COVID	6	4.6%
POST RSA-ABD. INJURY	1	0.8%
POST RSA-HEAD INJURY	2	1.5%
PROSTATOMEGALY	1	0.8%
RA	3	2.3%
SCLERODERMA	1	0.8%
SEIZURE DISORDER	1	0.8%
N	91	70%
Total	130	100.0%

In various other past medical histories post covid19 disease status was seen in 6/130 (4.6%) cases, depressive illness and rheumatoid arthritis 3/130 (2.3%) each, old pulmonary tuberculosis, parkinson disease, extrapulmonary tuberculosis and PLHIV 2/130(1.5%) each, CA prostate, bipolar disorder, bullous pemphigoid, dementia,hyperthyroidism, HCV reactive status, scleroderma 1/130(0.8%) each.

Table 7: Past surgical history

Surgical History	No. of cases	Percentage
Arthroplasty	1	0.8%
Below Knee Amputation	1	0.8%
Big Toe Amputation	1	0.8%
Cholecystectomy	2	1.5%
Cholecystectomy, Hernia Surgery	1	0.8%
D. Craniotomy, Post Tracheostomy	1	0.8%
DJ Stenting	1	0.8%
Foot Amputation	1	0.8%
Hysterectomy	1	0.8%
Post CABG	8	6.2%
Post CABG, Post TURP	1	0.8%
Post Craniectomy	1	0.8%
Post DJ Stenting	1	0.8%
Post Laparotomy	1	0.8%
Post LSCS	2	1.5%
Post Nephrectomy	1	0.8%
Post PTCA	6	4.6%
Post RSA Abd. Sx	1	0.8%
Post Tracheostomy	1	0.8%
Post TURBT	1	0.8%
Post TURP	1	0.8%
Total Knee Replacement	1	0.8%

8/130 (6.2%) cases had h/o CABG in the past, 6/130 cases had h/o PTCA. Other past surgical histories include Cholecystectomy, LSCS, amputations, TURP, TURBT, Tracheostomy, Hysterectomy, knee replacement, nephrectomy.

Table 8: Personal History

Personal history (Alcohol/ smoking/ other substances)	No. of cases	Percentage
Alcohol	33	25.4%
Smoking	21	16.2%
other substance (opioid, tobacco etc)	7	5.4%

33/130 (25.4%) had a history of alcohol consumption, 21/130 (16.2%) had history of smoking and 7/130 (5.4%) cases had history of other substance abuse.

Figure 6: Personal history.

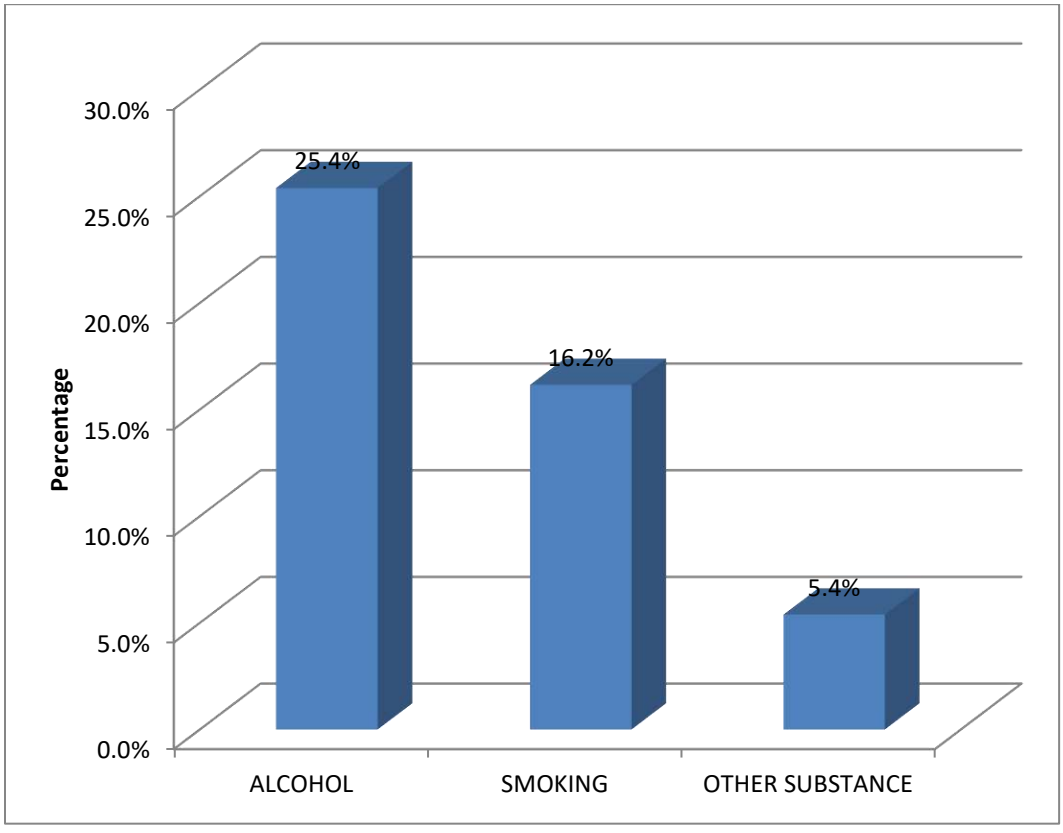


Table 9: Glasgow Coma Score (GCS) at Presentation

GCS	No. of cases	Percentage
< 7	27	20.8%
8-12.0	30	23.1%
13-15.0	73	56.2%
Total	130	100.0%

	n	Min	Max	Mean	SD
GCS	130	3.00	15.00	11.82	3.77

Maximum GCS was 15 and minimum score of 3 with mean of 11.82 and SD 3.77. 73/130 (56.2%) patients had GCS in range 13-15 and 27/130 (20.8%) patients had GCS <7.

Figure 7: Glasgow Coma Score (GCS) at Presentation

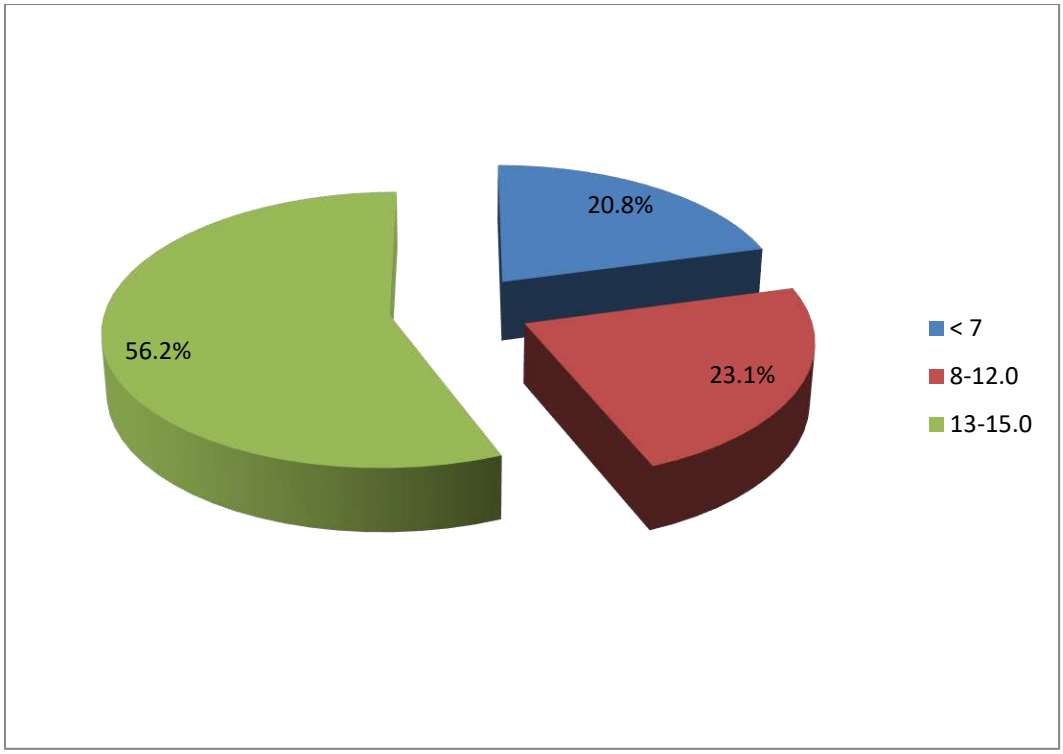


Table10: Vitals at Presentation

Vitals	n	Min.	Max.	Mean	SD
SBP(mmHg)	130	40.00	200.00	99.08	26.75
DBP(mmHg)	130	20.00	110.00	61.08	16.86
HR(beats/min)	130	68.00	164.00	107.46	18.88
RR(breaths per min)	130	12.00	46.00	23.75	5.56
SPO2%	130	50.00	100.00	88.45	10.43
FIO2 %	130	21.00	100.00	47.57	31.32

Maximum systolic blood pressure noted was 200mmHg and minimum SBP was 40mmHg with mean 99.08mmHg with SD of 26.75. Maximum Diastolic blood pressure was 110mmHg and minimum DBP was 20 mmHg with mean 61.08 and SD of 16.86. Maximum Heart rate was 164/min and Minimum HR noted was 68/min with mean of 107.46 and SD of 18.88. Maximum Resp. rate was 46/min and minimum 12/min with mean 23.75 and SD of 5.56. Maximum saturation (Spo2) was 100% and Minimum spo2 was 50% with mean value of 88.45% and SD of 10.43.

Table11: Lab Investigations at Presentation

Parameters	n	Min	Max	Mean	SD
TLC (X 1000/ UL)	130	3.20	40.60	14.79	7.00
Hb (gm/dl)	130	3.90	17.60	10.50	2.52
Platelets (X 1000/ UL)	130	6.00	783.00	215.37	127.81
Urea (mg/dl)	130	12.00	288.00	93.75	66.85
Creatinine (mg/dl)	130	0.37	8.80	2.70	2.09
S. Sodium (mmol/L)	130	8.20	174.00	135.49	14.27
S. Potassium (mmol/L)	130	2.66	7.35	4.53	0.98
S. Chloride (mmol/L)	130	69.00	136.00	100.45	8.89
S. Calcium	118	6.20	12.00	8.31	0.94
S. Magnesium	113	1.00	4.29	2.17	0.59
S. Phosphorus	114	0.56	14.00	4.32	2.25
Total Bilirubin(mg/dl)	130	0.04	18.30	1.22	2.02
SGOT (U/L)	130	9.00	2735.00	160.15	447.26
SGPT (U/L)	130	2.00	1696.00	98.35	232.41
ALP (U/L)	130	38.00	664.00	145.88	95.54
Total Protiens (gm/dl)	130	4.00	9.20	6.14	0.98
S. Albumin (gm/dl)	130	1.32	5.55	3.07	0.72
Urine Pus cell	60	2.00	100.00	21.88	28.17
ESR	111	2.00	145.00	51.35	34.65
CRP (mg/L)	73	0.68	412.00	111.90	101.83
PCT	120	0.05	100.00	11.84	22.68
B-1,3 GLUCAN ASSAY	43	7.80	523.00	177.95	153.88

Maximum Leucocyte count noted was 40.6 (x1000 /UL) and Minimum WBC count was 3.20 (x1000/UL) with mean of 14.79 and SD 7.0. Maximum Hemoglobin was 17.60gm/dL and Minimum Hb of 3.90gm/dL with mean Hb of 10.50 and SD of 2.52. Maximum platelet count noted was 783(x1000/UL) and minimum 6.00(x1000/UL) with mean of 215.37 and SD of 127.81. Maximum serum creatinine noted was 8.80 mg/dL and minimum 0.37 mg/dL with mean of 2.70 and SD of 2.09. Maximum serum potassium was

7.35mmol/L and minimum 2.66mmol/L with mean 4.53 and SD 0.98. Maximum serum Total Bilirubin was 18.30mg/dL and minimum 0.04mg/dL with mean 1.22mg/dL and SD of 2.02. Maximum SGOT noted was 2735U/L and Minimum 9 U/L with mean of 160.15 and SD 447.26. Maximum SGPT noted was 1696 U/L and Minimum 2 U/L with mean of 98.35 and SD of 232.41. Maximum Urine pus cells noted were 100/ HPF and Minimum 2 pus cells/ HPF with mean of 21.88 and SD of 28.17. Maximum ESR noted was 145 and minimum was 2 with mean of 51.35 and SD 34.65. Maximum CRP-Q was 412mg/L and minimum value of 0.68 mg/l with mean of 111.90 and SD 101.83. Maximum PCT value noticed was 100 ng/mL and minimum value of 0.05 ng/mL with mean of 11.84 and SD of 22.68.

Table 12: Viral Markers

HBV/ HCV/ HIV STATUS	No. of cases	Percentage
HBV REACTIVE	1	0.8%
HCV REACTIVE	5	3.8%
HCV, HIV REACTIVE	1	0.8%
HIV REACTIVE	1	0.8%
NR	79	60.8%
-	43	33.1%
Total	87	100.0%

(- means not tested)

5/87 (3.8%) cases were HCV positive, 1/87 (0.08%) was HBV positive and HIV positive each. 1/87 (0.08%) cases were HIV and HCV both positive. 79/130 cases (60.8%) are Non Reactive. Status of 43/130 cases (33.1%) was not tested.

Figure 8: Viral Markers

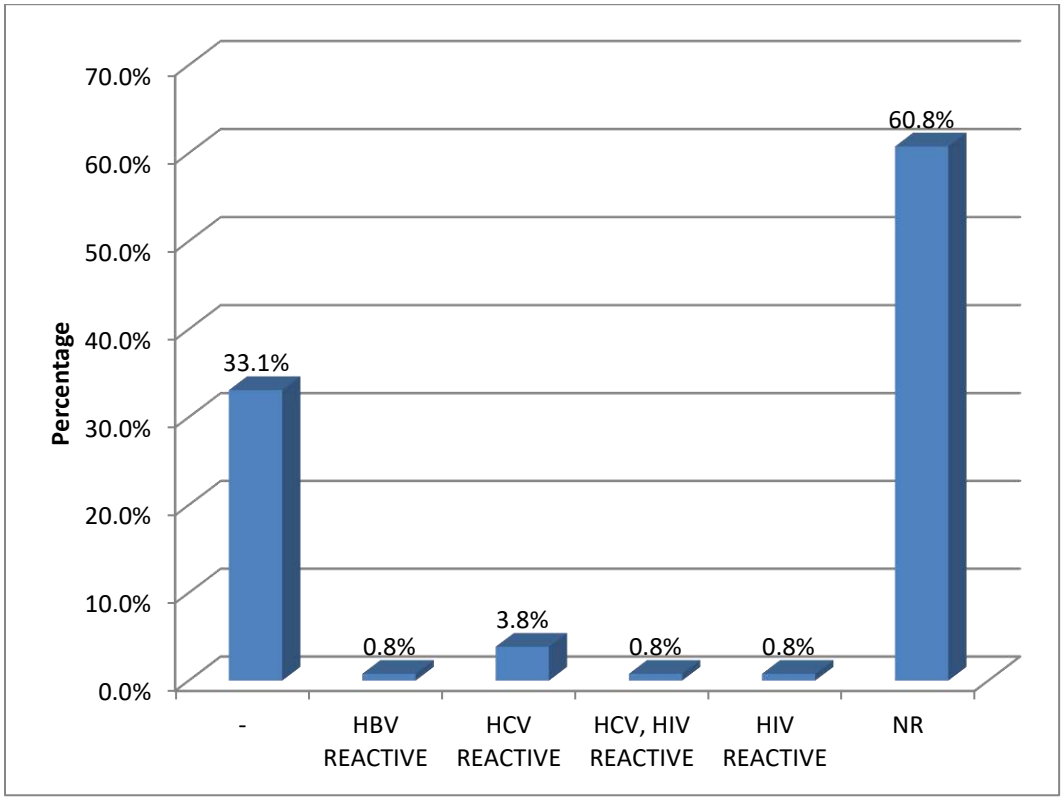


Table 13: Cases with Culture Growth isolation

Culture Growth	n	No. of cases	Percentage
Urine	100	33	33.0%
Blood	92	24	26.1%
CSF	7	0	0.0%
Sputum/ET Secretion	57	33	57.9%
Pus/Wound Swab	14	8	57.1%
Pleural fluid	2	1	50.0%
Ascitic fluid	5	1	20.0%

33/100 (33%) of Urine cultures had growths in them, 33/57 (57.9%) Sputum/ ET secretion cultures had growth in them and 8/14 (57.1%) of Pus/Wound swab cultures had positive growth in them. The rest of the identified infective foci were blood, CSF, pleural fluid and intra abdominal infections.

Figure 9: Cases with Culture Growth isolation

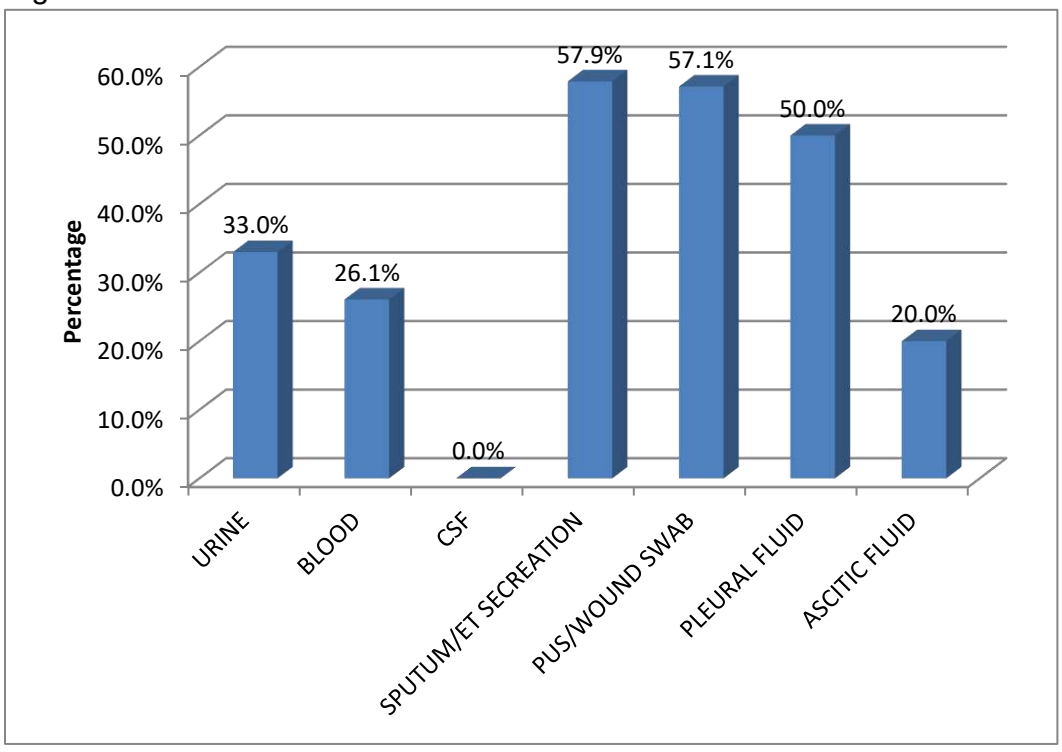


Table 14: Site of Infection

Site of infection	No. of cases	Percentage
RTI	76	58.5%
UTI	60	46.2%
GIT	12	9.2%
Skin	14	10.8%
Blood Stream	25	19.2%
CNS infection	4	3.1%
Genital infection	4	3.1%

Most common source of infection was respiratory tract in 76/130 (58.5%) patients followed by urinary tract 60/130 (46.2%) patients, 25/130 (19.2%) had blood stream infection, 14/130 (10.8%) had skin infection, 12/130 (9.2%) had GIT infection, CNS infection and Genital infection was present in 4/130 (3.1%) patients each.

Figure 10: Site of Infection

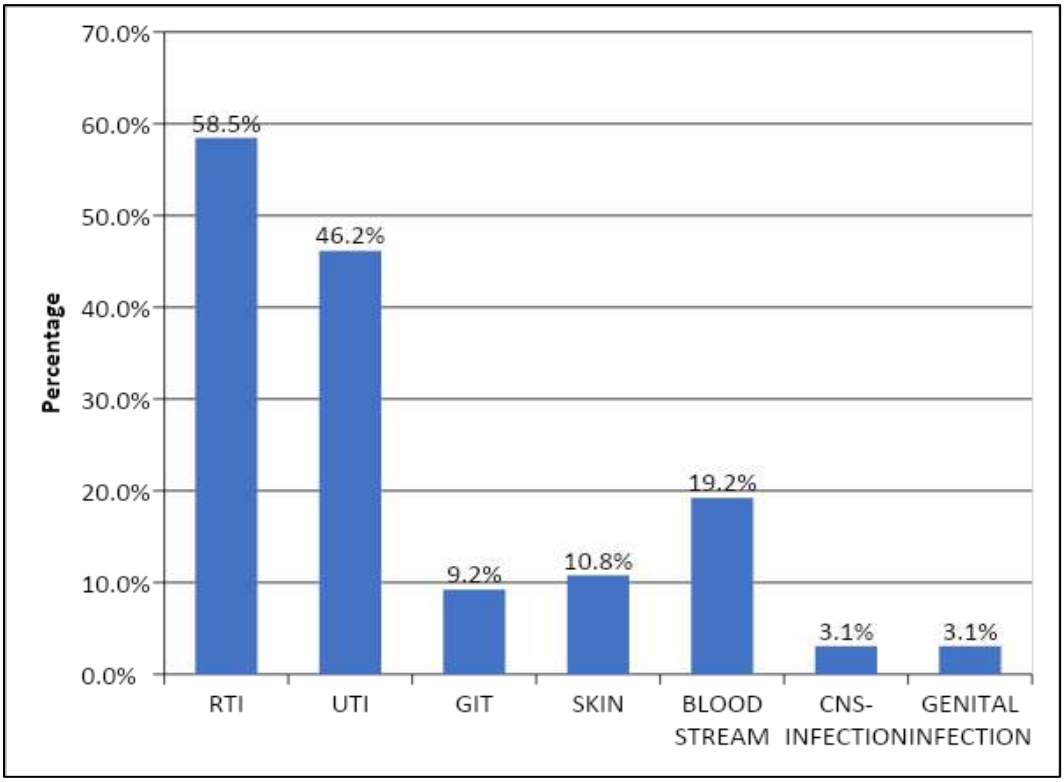


Table 15: Causative Agent of UTI

Urine Culture Growth	No. of cases	Percentage
Candida	12	12.0%
E.Coli	5	5.0%
Enterococcus	4	4.0%
Klebsiella	6	6.0%
Morganella	1	1.0%
Neg	67	67.0%
Proteus	1	1.0%
Providencia	2	2.0%
Pseudomonas	5	5.0%

(n=100)

Most common causative agent of UTI was Candida with 12/100 (12%)cases, Klebsiella 6/100 (6%) cases, Pseudomonas and E.Coli 5/100 (5%) cases each. Other bacterial isolated were Enterococcus, Providencia,Proteus, Morganella.

Figure 11: Causative agent of UTI

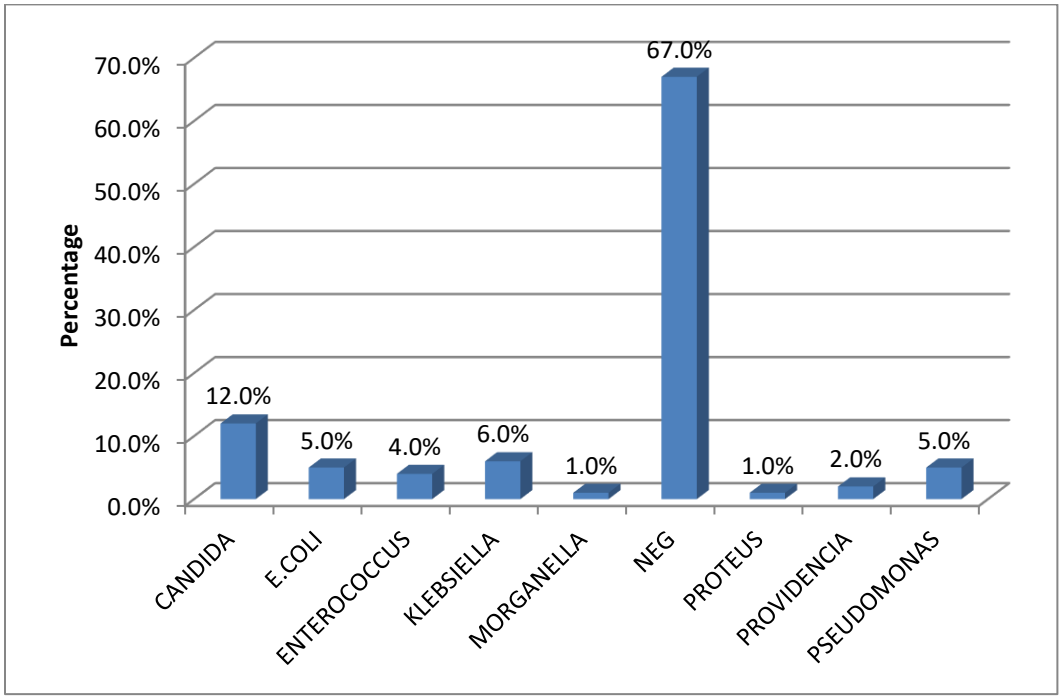


Table 16: Causative agent of Bacteremia

Culture Growth	No. Of Cases	Percentage
Candida	2	2.2%
Citrobacter	1	1.1%
E.Coli	5	5.4%
Enterococcus	1	1.1%
Klebsiella	8	8.7%
Enterobacter	1	1.1%
Morganella	1	1.1%
Neg	68	73.9%
Pseudomonas	1	1.1%
Serratia Marces.	1	1.1%
Staph epidermidis	4	4.3%
Staph hemolyticus	1	1.1%

(N=92)

Most Common Cause Of Bacteremia Was Klebsiella 8/92 (8.7%) Cases, E.Coli 5/92 (5.4%) And Staph. epidermidis 4/92 (4.3%) Cases. Other Causes Of Bacteremia Was Candida, Citrobacter, Enterococcus, Enterbacter, Morganella, Serratia marcescens, And Staph. hemolyticus

Figure 12: Causative Agent of Bacteremia

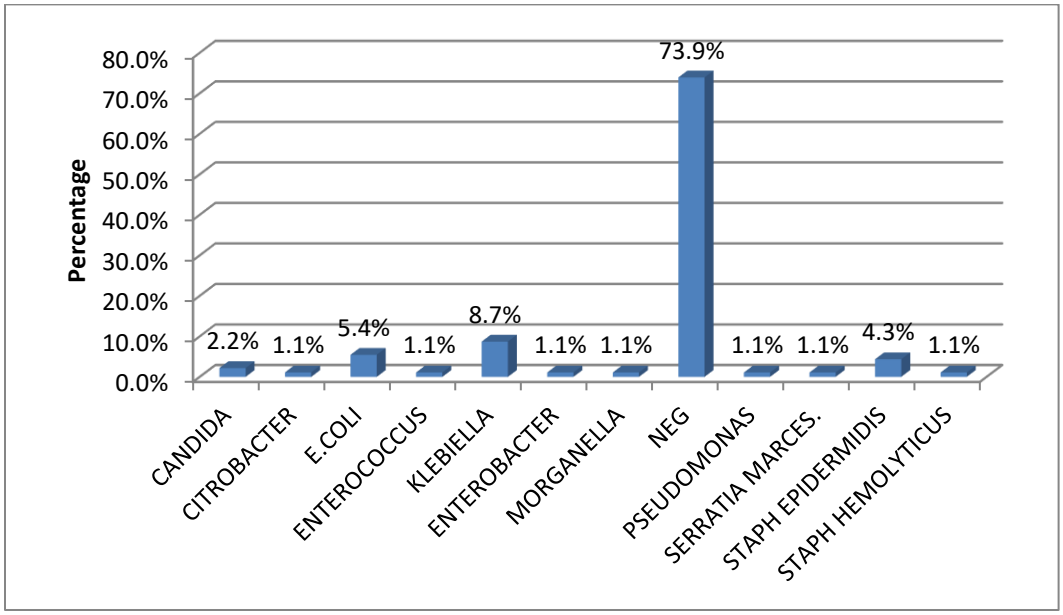


Table 17: Causative Agent of LRTI

Sputum/ET Secretion Culture Growth	No. of cases	Percentage
Acinetobacter	19	33.3%
Proteus	1	1.8%
Candida	2	3.5%
E.Coli	2	3.5%
Klebsiella	18	31.6%
Aspergillus	1	1.8%
Neg	24	42.1%
Pseudomonas	1	1.8%
Serratia	1	1.8%

(n=57)

Most common organism isolated was Acinbetobacter in 19/57 (33.3%) and Klebsiella in 18/57 (31.6%). Other organism isolated were Candida, E.Coli, Proteus, Aspergillus, Pseudomonas and Serratia.

Figure 13: Causative agent of LRTI

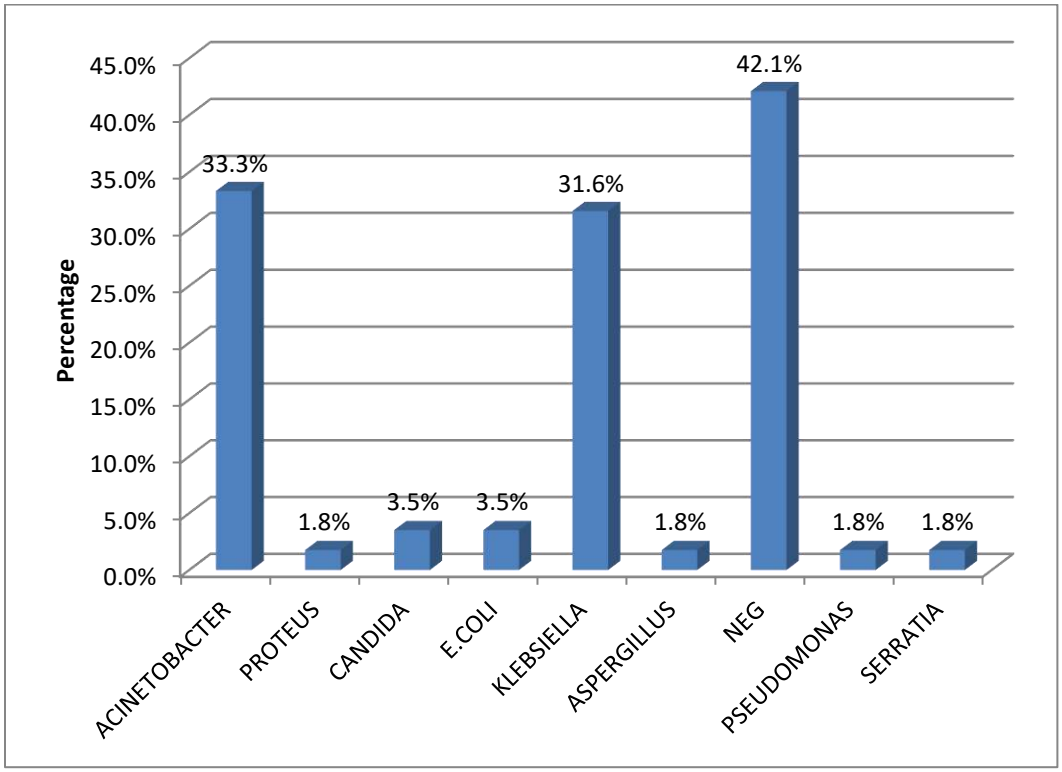


Table 18: Causative Agent of Soft Tissue Infection

Pus/Wound Swab Culture Growth	No. of cases	Percentage
Acinetobacter	3	21.4%
Citrobacter	1	7.1%
E.Coli	1	7.1%
Klebsiella	5	35.7%
Pseudomonas	2	14.3%
Neg	6	42.9%

(n=14)

Most common cause of Soft Tissue infection was Klebsiella in 5/14 (35.7%) cases followed by Acinetobacter 3/14 (21.4%) cases and Pseudomonas 2/14 (14.3%) cases.

Figure 14: Causative Agent of Soft Tissue Infection

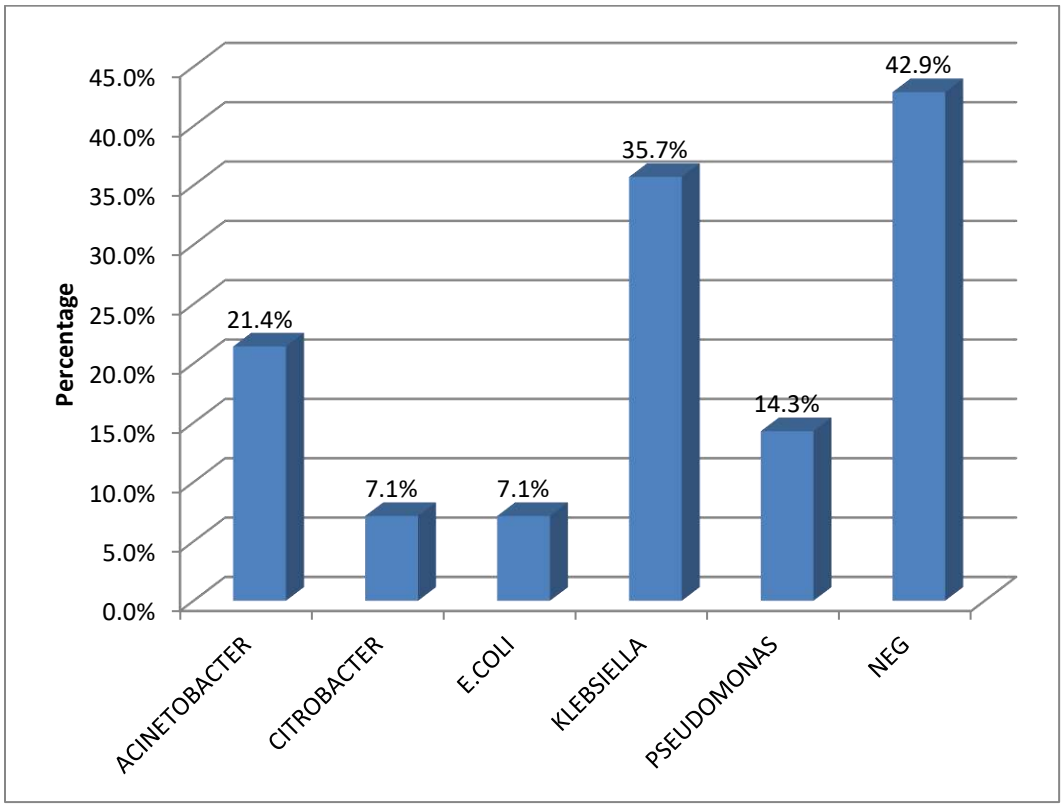


Table 19: Causative agent of Peritonitis

ASCITIC FLUID	No. of cases	Percentage
E.COLI	1	20.0%
NEG	4	80.0%
Total	5	100.0%

(n=5)

E.Coli was isolated in 1/ 5 cases (20%) from Ascitic fluid culture.

Figure 15: Causative agent of Peritonitis

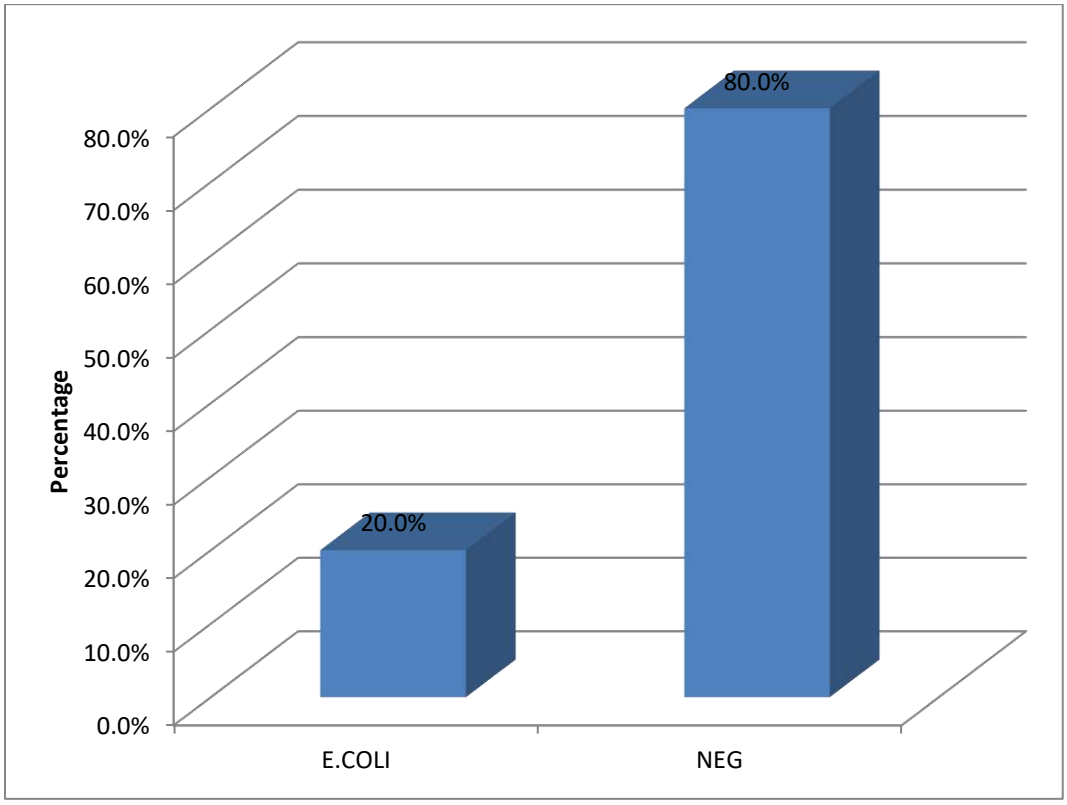


Table 20: SOFA Score

	n	Min	Max	Mean	SD
SOFA SCORE ON INCLUSION DATE	130	2.00	20.00	9.76	3.88

Minimum SOFA score noted in our study was 2 and Maximum SOFA score was 20 with Mean SOFA of 9.76 and SD of 3.88

Table 21: Left ventricular ejection fraction (LVEF) on 2D-ECHO.

LVEF **%**	No. of cases	Percentage
< 20	8	6.2%
20-40	96	73.8%
41-50	26	20.0%
Total	130	100.0%

96/130 (73.8%) cases had LVEF between 20-40%, 26/130 (20.0%) had LVEF 41-50% and 8/130 (6.2%) had LVEF < 20%.

Figure 16: Left ventricular ejection fraction (LVEF) on 2D-ECHO

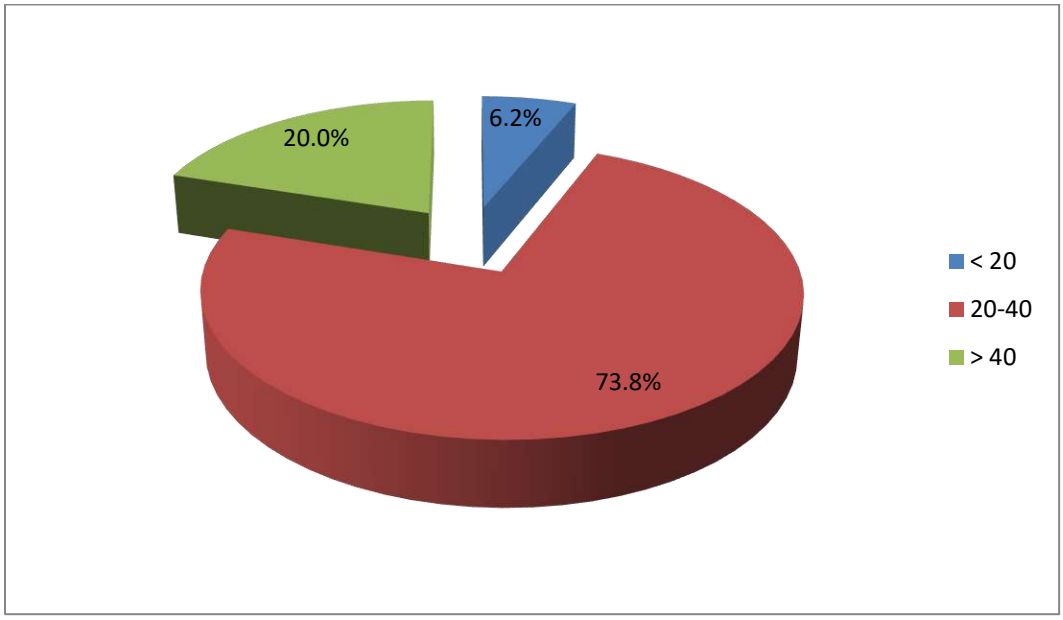


Table 22: Ventricular Global Hypokinesia (GHK)/ Regional wall motion abnormality (RWMA)

GHK/RWMA	No. of cases	Percentage
GHK	84	64.6%
RWMA	46	35.4%
Total	130	100.0%

84/130 (64.6%) cases had global hypokinesia in 2D ECHO and 46/130 (35.4%) cases had regional wall motion abnormality on 2D ECHO.

Figure 17: Ventricular Global Hypokinesia (GHK)/ Regional wall motion abnormality (RWMA)

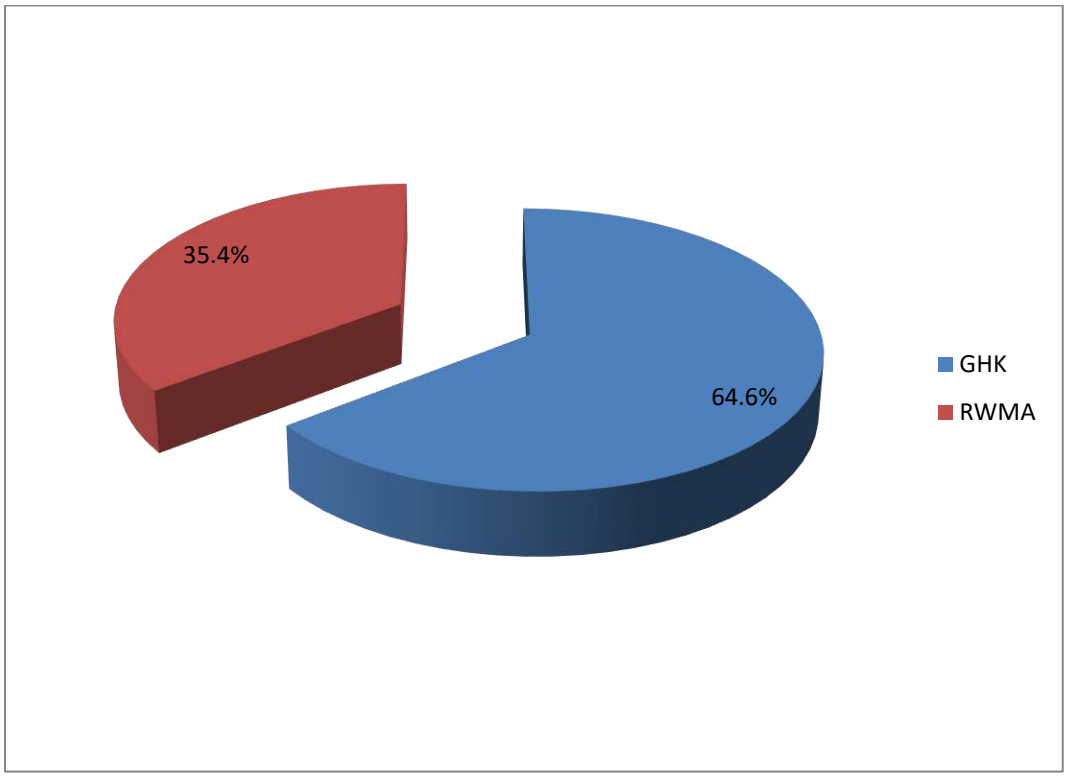


Table 23: Outcome of cases Admitted with Sepsis and LVEF<50%

OUTCOME	No. of cases	Percentage
DAMA	44	33.8%
DEATH	20	15.4%
DISCHARGE	66	50.8%
Total	130	100.0%

66/130 (50.8%) cases were discharged from hospital, 44/130 (33.8%) discharged against medical advice and 20/130 (15.4%) expired during treatment.

Figure 18: Outcome of cases Admitted with Sepsis AND LVEF<50%

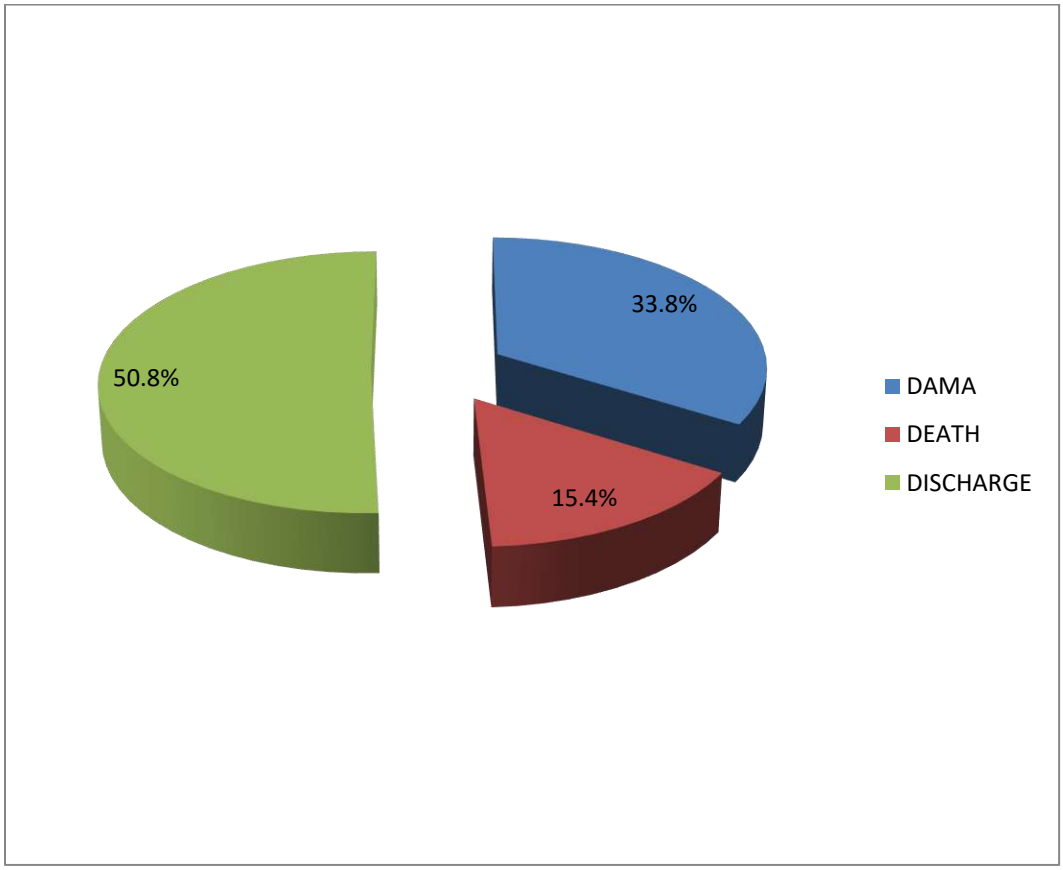


Table 24: Total Hospital Stay Duration of cases

	cases	Min	Max	Mean	SD
TOTAL HOSPITAL STAY DURATION (DAYS)	130	2.00	51.00	14.22	9.10

Minimum hospital stay noted was 2 days and Maximum hospital stay noted was 51 days with Mean of 14.22 and SD of 9.10

Table 25: 30 day Mortality Outcome

Status Of Patient At 30 Days After Discharge	No. of cases	Percentage
DEAD	66	50.8%
ALIVE	64	49.2%
Total	130	100.0%

66/130 (50.8%) cases expired within 30 days of discharge from hospital and 64/130 cases (49.2%) were alive 30 days after discharge.

Figure 19: 30 day Mortality Outcome

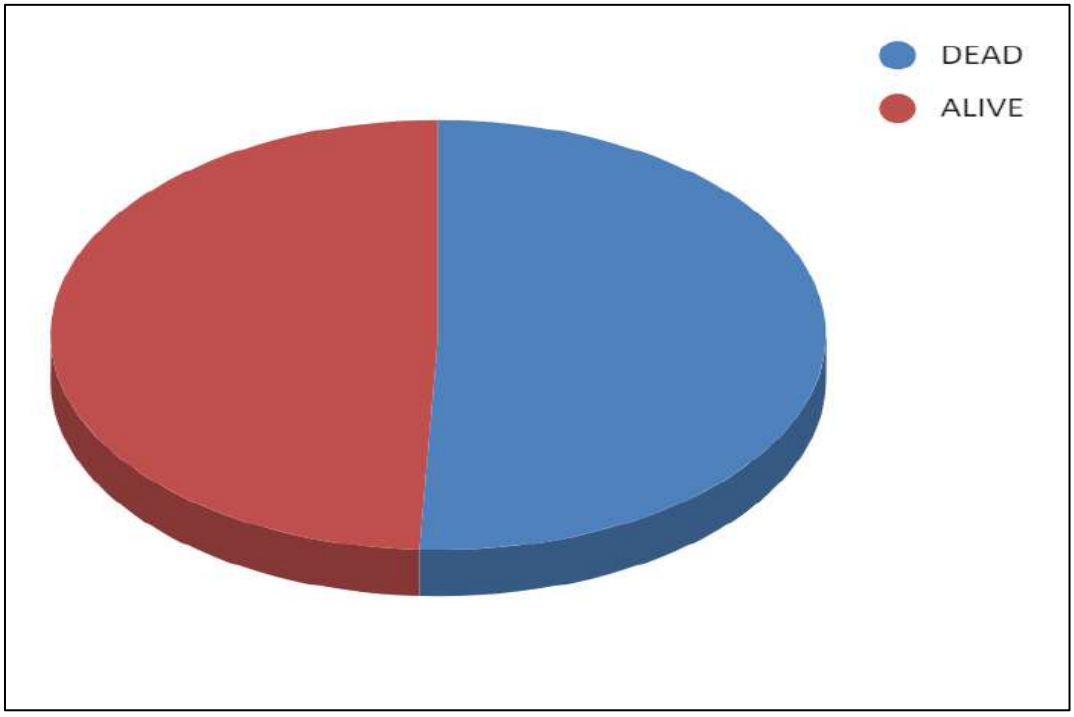


Table26: Association of Age with 30 day Mortality

		DEAD				Total	Chi-square value	p-value
		N	N	Y	Y			
AGE GROUP	< 30	6	46.2%	7	53.8%	13	5.401	0.369
	31-40	5	55.6%	4	44.4%	9		
	41-50	10	71.4%	4	28.6%	14		
	51-60	17	54.8%	14	45.2%	31		
	61-70	17	44.7%	21	55.3%	38		
	> 70	9	36.0%	16	64.0%	25		
Total		64	49.2%	66	50.8%	130		

Out of a total 130, 66 patients expired at 30 days of discharge. Maximum expired patients were in the age group >70yr and 61-70yr group with p value 0.369 which is statistically non-significant.

Figure20: Association of Age with 30 day Mortality in patients with Sepsis and LV systolic dysfunction <50%

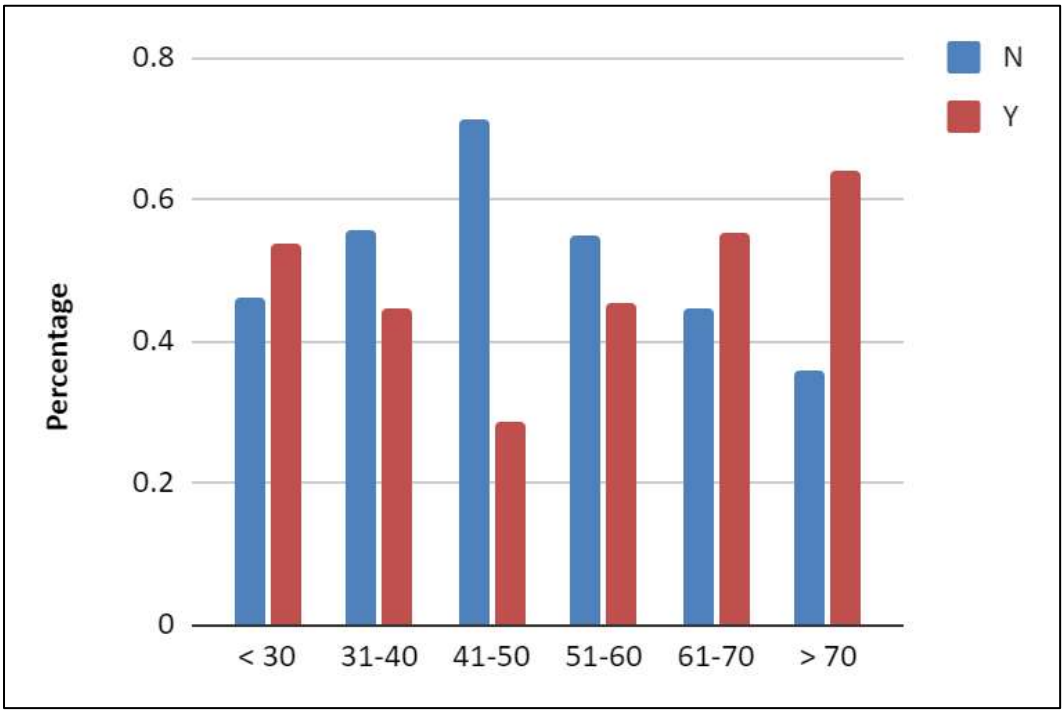


Table 27: Association of Gender with 30 day Mortality

		DEAD				Total	Chi-square value	p-value
		N	N	Y	Y			
SEX	F	21	46.7%	24	53.3%	45	0.181	0.670
	M	43	50.6%	42	49.4%	85		
Total		64	49.2%	66	50.8%	130		

Out of 66/130 expired patients, 42/66 were males and 24/66 were females with p value 0.670 which is statistically insignificant.

Figure 21: Association of Gender with 30 day Mortality.

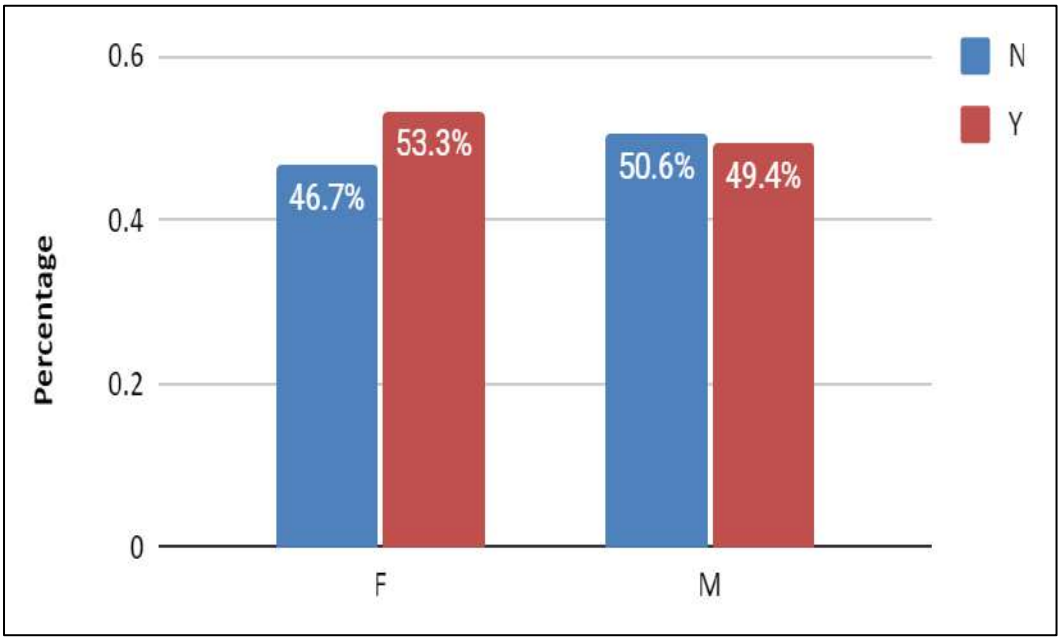


Table 28: Association of Presenting complaints with 30-Day mortality.

		DEAD				Total	Chi-square value	p-value
		N	N	Y	Y			
FEVER	Y	31	46.3%	36	53.7%	67	0.485	0.486
COUGH/SPUTUM	Y	24	63.2%	14	36.8%	38	4.167	0.054
PAIN ABDOMEN	Y	9	60.0%	6	40.0%	15	0.787	0.421
DECREASED URINE OUTPUT	Y	11	32.4%	23	67.6%	34	5.247	0.028
BURNING/ INCREASED FREQUENCY OF URINATION	Y	4	30.8%	9	69.2%	13	1.97	0.242
LOOSE STOOLS	Y	0	0.0%	5	100.0%	5	5.042	0.058
ALTERED SENSORIUM/ SEIZURES	Y	17	35.4%	31	64.6%	48	5.81	0.019
SOB	Y	33	54.1%	28	45.9%	61	1.089	0.380
VOMITING/NAUSEA	Y	8	72.7%	3	27.3%	11	2.654	0.124

36/67 (53.47%) cases presented with fever were expired with p value 0.486 which was statistically insignificant. 23/34 (67.6%) cases presented with decreased urine output expired with p value 0.028 which was statistically significant. 31/48 (64.6%) patients presented with altered sensorium expired with p value 0.019 which was statistically significant.

Figure 22: Association of Presenting complaints with 30-Day mortality.

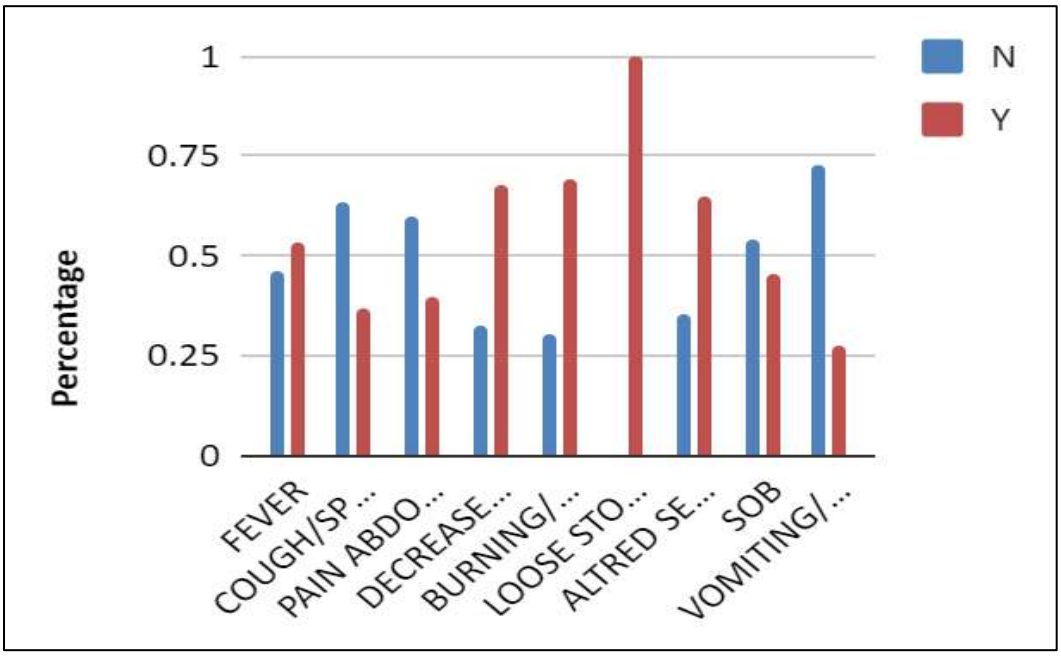


Table29: Association of comorbidities with Mortality

		DEAD				Total	Chi-square value	p-value
		N	N	Y	Y			
DM	Y	31	41.9%	43	58.1%	74	3.702	0.076
HTN	Y	30	43.5%	39	56.5%	69	1.947	0.218
COPD/B.ASTHMA/ ANY RESP DISORDER	Y	7	58.3%	5	41.7%	12	0.438	0.508
CLD	Y	2	33.3%	4	66.7%	6	0.636	0.680
CKD	Y	5	25.0%	15	75.0%	20	5.552	0.027
CAD	Y	15	42.9%	20	57.1%	35	0.778	0.432
HYPOTHYROIDISM	Y	7	46.7%	8	53.3%	15	0.045	0.833
CVA	Y	1	12.5%	7	87.5%	8	4.601	0.062

Patients were having various comorbid ailments out of which major were diabetes mellitus, hypertension, COPD/Bronchial Asthma, Chronic Liver Disease, Chronic kidney Disease, CAD, Hypothyroidism, and CVA. 15/20 patients of CKD expired with p value 0.027 which was statistically significant.

Figure 23: Association of comorbidities with Mortality

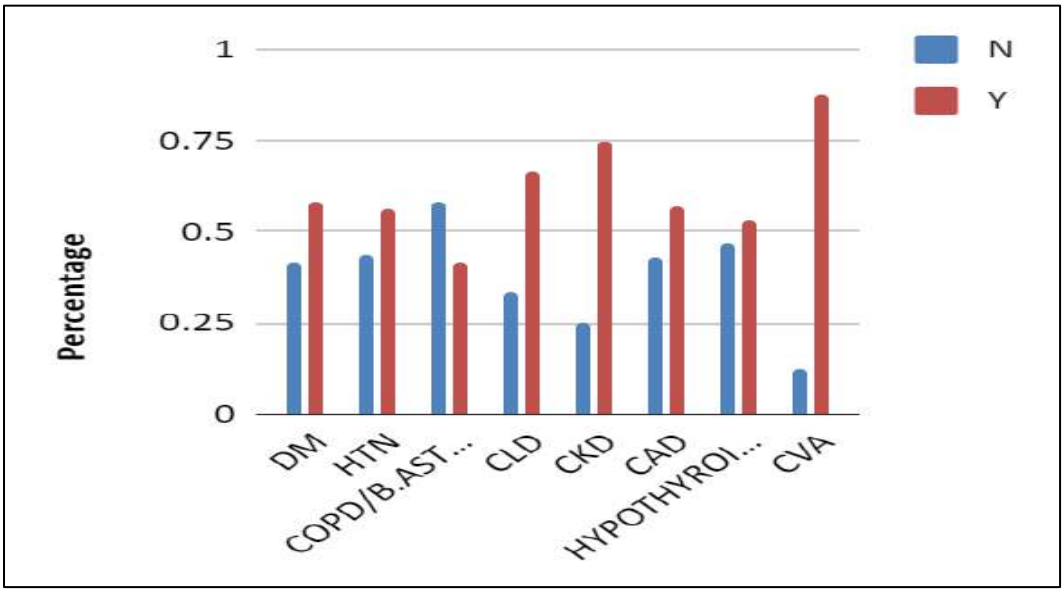


Table 30: Association of presenting GCS with 30 day mortality

		DEAD				Total	Chi-square value	p-value
		N	N	Y	Y			
gcs GROUP	< 7	9	33.3%	18	66.7%	27	6.486	0.039
	8-12.0	12	40.0%	18	60.0%	30		
	13-15.0	43	58.9%	30	41.1%	73		
Total		64	49.2%	66	50.8%	130		

GCS of <7 has highest association with mortality with 18/27 (66.7%) mortalities and p value of 0.039 which is statistically significant. Among GCS group of 13-15, only 30/73 (41%) cases expired. Low GCS <7% is associated with worse outcome.

Figure24: Association of presenting GCS with 30 day mortality

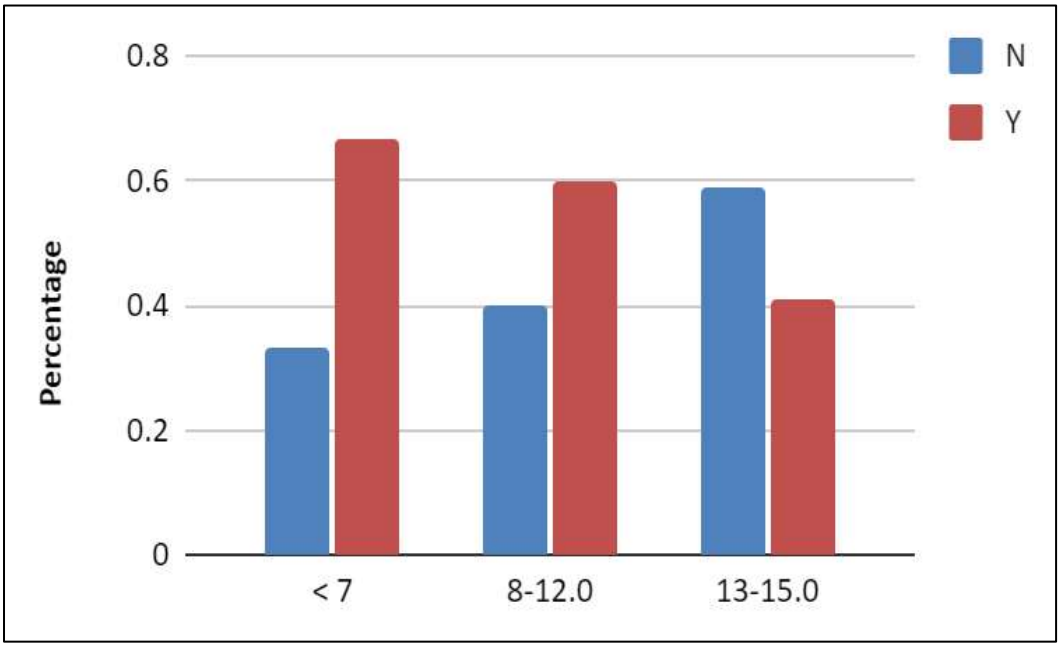


Table31: Association of substance use with mortality

		DEAD				Total	Chi-square value	p-value
		N	N	Y	Y			
ALCOHOL	N	46	47.4%	51	52.6%	97	0.500	0.548
	Y	18	54.5%	15	45.5%	33		
SMOKING	N	54	49.5%	55	50.5%	109	0.026	0.872
	Y	10	47.6%	11	52.4%	21		
OTHER SUBSTANCE	N	60	48.8%	63	51.2%	123	4.043	0.400
	Y	4	80.0%	3	60.0%	5		

Out of 33/130 cases with history of alcohol consumption in past 54.5% cases survived. Out of 97/130 cases with no history of alcohol consumption, 52.6% cases expired with p value of 0.548 which is statistically insignificant. Out of 21/130 cases with h/o smoking in past 52.4% cases expired and 55/109 cases (50.5%), with no h/o smoking in past, survived with p value of 0.872 which is statistically insignificant

Figure 25: Association of substance use with mortality

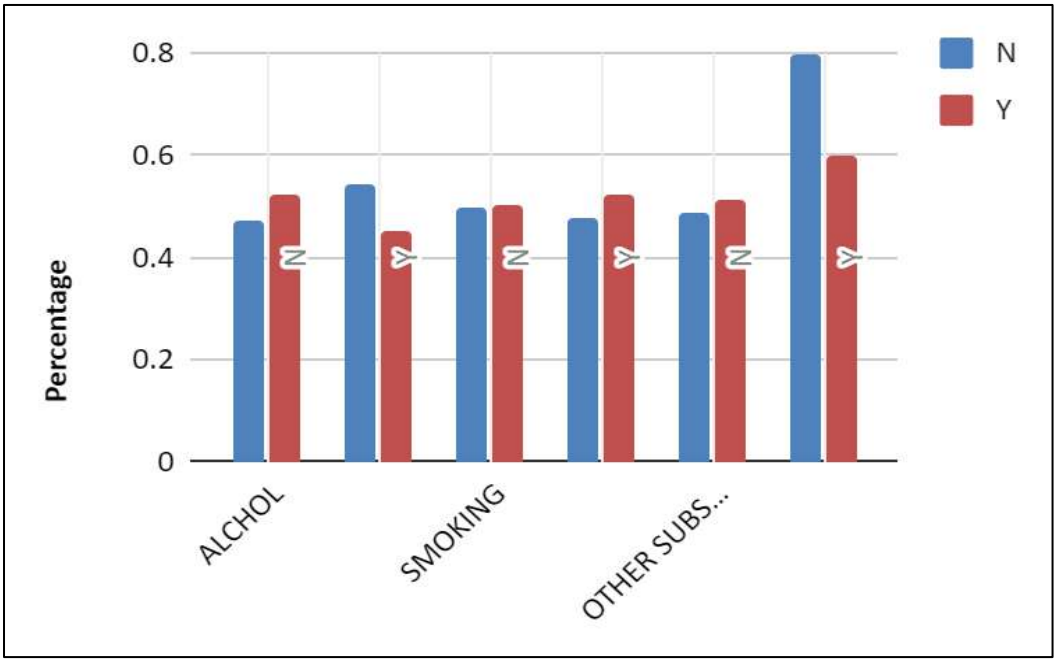


Table32: Association between site of infection and 30 day mortality.

		DEAD				Total	Chi-square value	p-value
		N	N	Y	Y			
RTI	Y	37	48.7%	39	51.3%	76	0.022	0.882
UTI	Y	25	41.7%	35	58.3%	60	2.551	0.118
GIT	Y	3	25.0%	9	75.0%	12	3.106	0.128
SKIN	Y	8	57.1%	6	42.9%	14	0.393	0.582
BLOOD STREAM	Y	12	48.0%	13	52.0%	25	0.019	0.891
CNS-INFECTION	Y	2	50.0%	2	50.0%	4	0.001	0.975
GENITAL INFECTION	Y	1	25.0%	3	75.0%	4	0.969	0.325

GIT infection with 9/12 (75%) mortalities and genital infections with 3/ 4 (75%)mortalities has the highest association with 30 day mortality with p value of 0.128 and 0.325 respectively. Then UTI with 35/60 (58.3%) mortalities has high association with 30 day mortality.

Figure26: Association between site of infection and 30 day mortality.

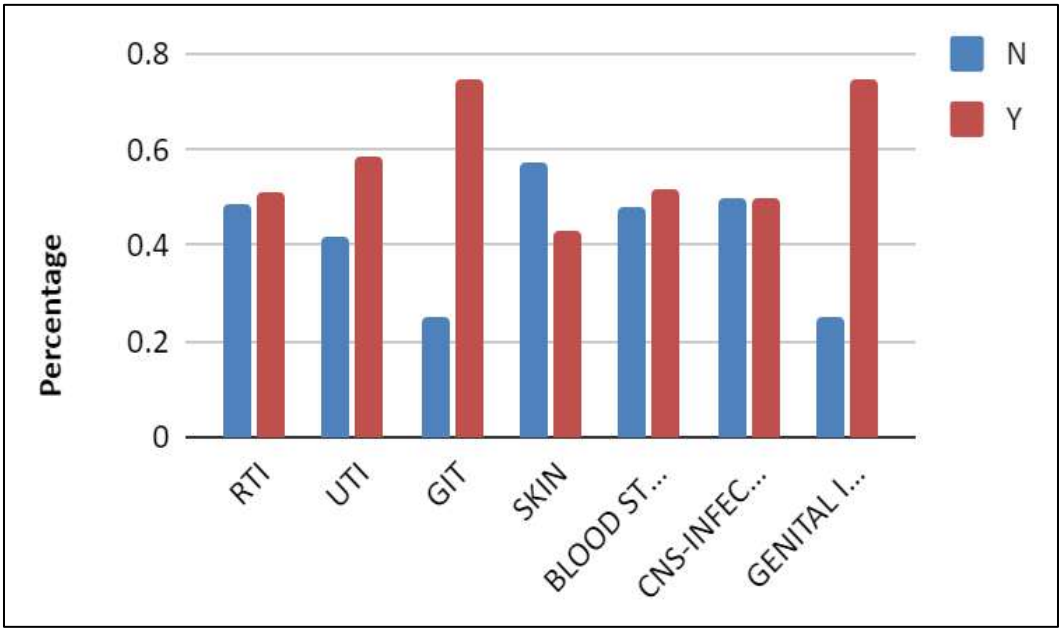


Table33: Association of Mean SOFA score with 30 day mortality

Dead	N	Y
	Mean	Mean
SOFA SCORE ON INCLUSION DATE	8.13	11.35

Higher Mean SOFA (11.35) was noted in cases who expired (30day mortality) and lower Mean SOFA (8.13) was noted in cases who were alive at 30 days

Table34: Association of SOFA score with 30 day mortality

		DEAD				Total	Chi-square value	p-value
		N	N	Y	Y			
SOFA	0-8.0	39	76.5%	12	23.5%	51	25.498	0.001
	9-16.0	1	16.7%	5	83.3%	6		
	17-24.0	24	32.9%	49	67.1%	73		
Total		64	49.2%	66	50.8%	130		

With SOFA score <8.0, 12/51 cases (23.5%) expired and 39/51 (76.5%) cases survived. With SOFA of 9-16, 5/ 6(83.3%) cases expired. Worse outcome is seen in cases with SOFA of >17 with 49/73 (67.1%) mortalities with p value of 0.001 which is statistically significant.

Figure 27: Association of SOFA score with 30 day mortality

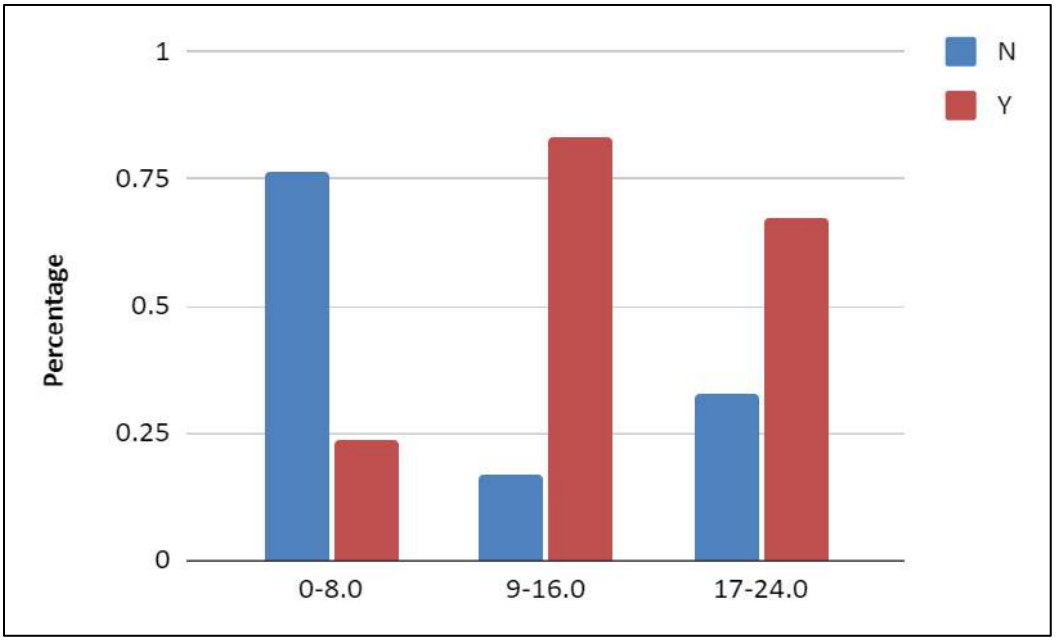


Table 35: Association of LVEF% with 30 day mortality

		DEAD				Total	Chi-square value	p-value
		N	N	Y	Y			
EF	< 20	3	37.5%	5	62.5%	8	1.252	0.535
	20-40	46	47.9%	50	52.1%	96		
	> 40	15	57.7%	11	42.3%	26		
Total		64	49.2%	66	50.8%	130		

In this study LVEF <20% was associated with highest mortality [5/ 8 cases (62.5%)] with p value of 0.535. With LVEF >40% mortality outcome is least [11/26 (42.3%) cases. This signifies a worsening outcome with worsening LVEF%.

Figure 28: Association of LVEF% with 30 day mortality

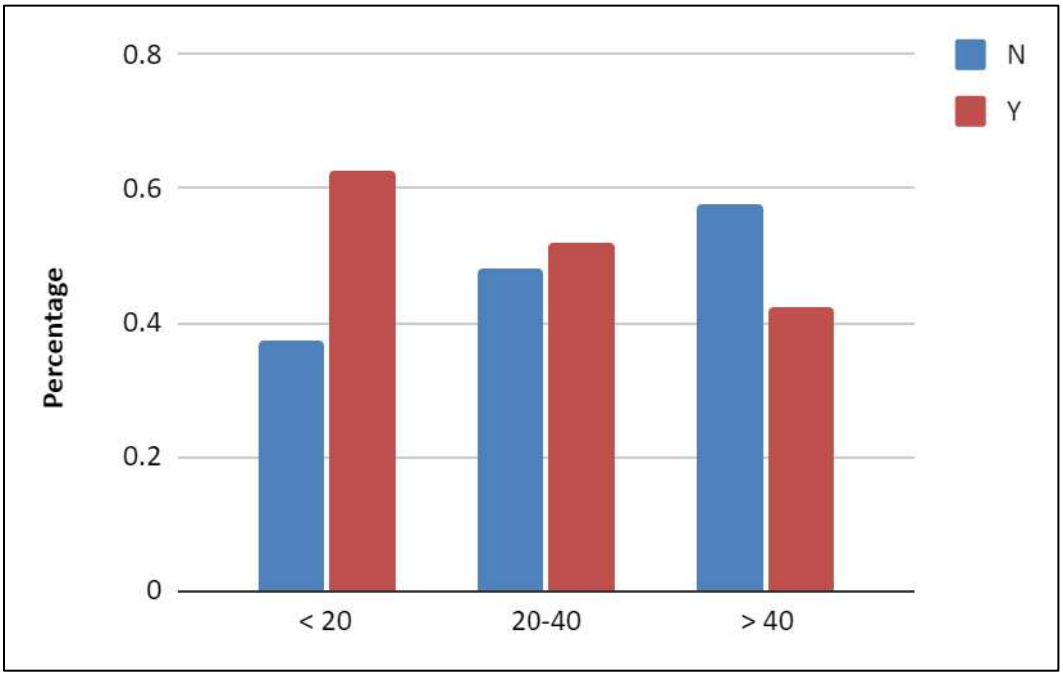


Table 36: Association of ventricular GHK/ RWMA with 30 day mortality

		DEAD				Total	Chi-square value	p-value
		N	N	Y	Y			
GHK/RWMA	GHK	42	50.0%	42	50.0%	84	0.056	0.813
	RWMA	22	47.8%	24	52.2%	46		
Total		64	49.2%	66	50.8%	130		

With 24/46 (52.2%) cases RWMA was shown to be more associated with mortality with p value of 0.813 which is statistically insignificant.

Figure 29: Association of ventricular GHK/ RWMA with 30 day mortality

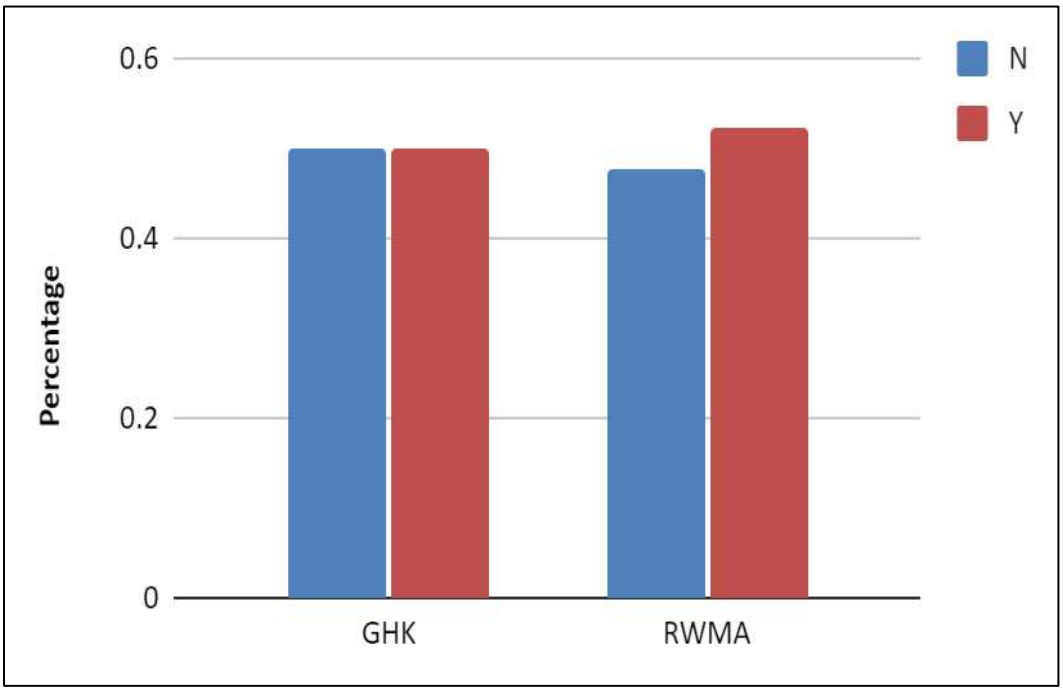


Table37: Association of Discharge/ DAMA status with 30 day Mortality

		DEAD				Total	Chi-square value	p-value
		N	N	Y	Y			
OUTCOME	DAMA	9	20.5%	35	79.5%	44	64.682	0.001
	DISCHARGE	55	83.3%	11	16.7%	66		
Total		64	58.2%	46	41.8%	110		

With 55/66 (83.3%) Discharged patients had better outcome and 30 day survival. With 35/44 (79.5%) cases DAMA patients had worse outcome/ 30 day mortality with p value of 0.001 which is statistically significant.

Figure 30: Association of Discharge/ DAMA status with 30 day Mortality

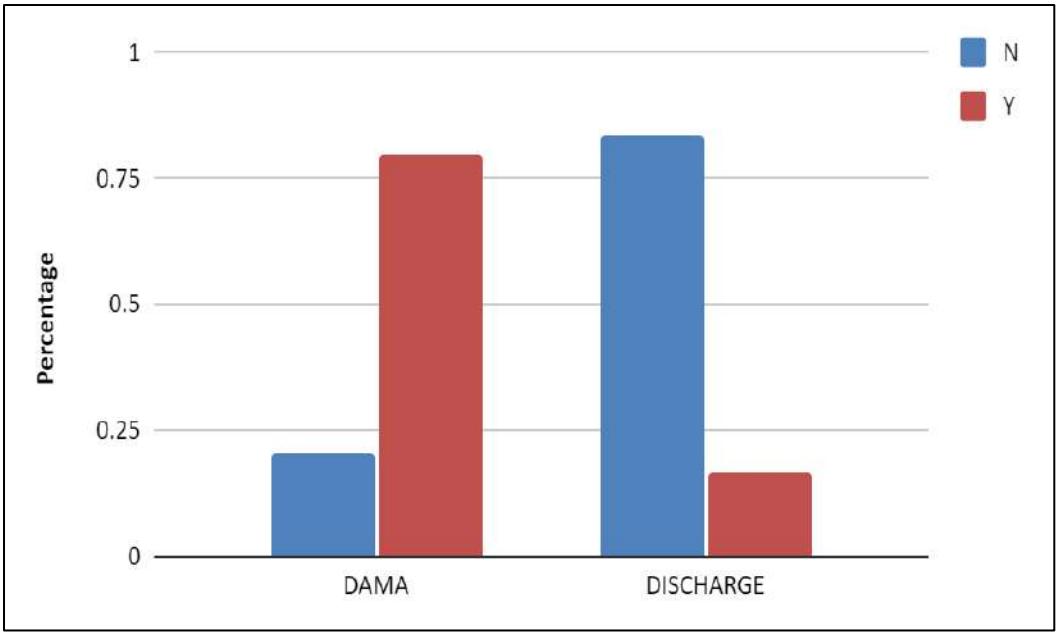
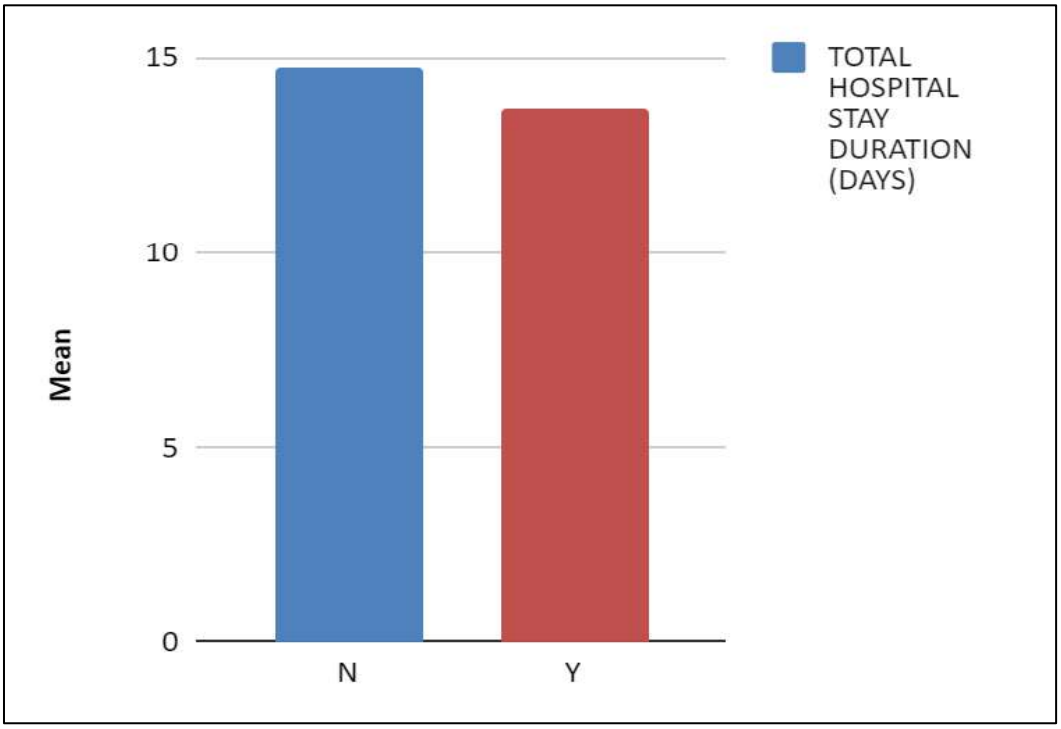


Table 38: Association of Total Hospital Stay Duration with 30 day Mortality

Dead	N	Y
	Mean	Mean
TOTAL HOSPITAL STAY DURATION (DAYS)	14.75	13.70

Mean Hospital Stay duration in survival group and expired group is 14.75 and 13.70 respectively.

Figure 31: Association of Total Hospital Stay Duration with 30 day Mortality



DISCUSSION

A prospective, single center ,observational study was conducted in medical ICU/HDU of Dayanand medical college and hospital , Ludhiana from march 1, 2021 to march 1, 2022. We aimed to study the clinical profile and mortality outcome of patients admitted to ICU/HDU with sepsis and Left ventricular systolic dysfunction. after following sepsis-3 guidelines, sepsis patients were evaluated for left ventricular systolic dysfunction using 2d-echocardiography. Patients having left ventricular ejection fraction (LVEF) <50% were included in the study. We studied the clinical profile i.e presenting symptoms, past medical/surgical history, vitals, SOFA score, lab investigations, culture growth reports, sepsis biomarkers(esr, crp, procalcitonin), 2d-echocardiography report, hospital stay duration, outcome (discharge/ dama/ death) and finally 30 day mortality of the patient.

STUDY POPULATION

a) Number of patients

The present study consisted of 130 patients between the ages of 18-100 years with maximum patients in age group 61-70 yr (29.2%) and minimum patients in age group 31-40yrs (6.9%). Our patients were relatively young with a mean age of 57.28 yr as compared to previous studies conducted by Simon F. et.al. (2004) [93] (mean age 60.7yr) and by Engel C. (2007) (67 yr)[94].

b) Gender

In our study, 45/130 (34.6%) females and 85/130 (65.4%) males were included . male predominance in our study was at par with the study conducted by TTS Paary et. al (2016) [80] (males 64.2%) and C.M.Raibhoge et.al. (2021) [87] (males 57.8% with male to female ratio 1.378:1)

Presenting complaints/ symptoms

Patient presented with fever 67/130 (51.5%), shortness of breath 61/130 (46.9%), then altered sensorium/ seizure 48/130 (36.9%), then cough/sputum production 38/130 (29.2%), followed by decreased urine

output 34/130 (26.2%), then pain abdomen 15/130 (11.5%), burning/ increased frequency of urination 13/130 (10%), then nausea/vomiting 11/130 (8.5%) cases. Study done by C.M.Raibhoge et.al.[87] (2021) had 100% of patients with fever, 67.2% patients having nausea/ vomiting as presenting symptoms. Various other associated presenting symptoms were chest pain, lethargy/malaise, pedal swelling, abdomen distension, blood in sputum, jaundice, loss of appetite, limb weakness, and snake bite, opc poisoning.

Comorbidities

In our study 74/130 (56.9%) patients were having Diabetes Mellitus, 69/130 (53.1%) were having hypertension, 35/130 (26.9%) had coronary artery disease, 20/130 (15.4%) had chronic kidney disease, 15/130(11.5%) had hypothyroidism, 12/130 (9.2%) had chronic respiratory illness, 8/130 (6.2%) had history of cerebrovascular accident, 6/130 (4.6%) had chronic liver disease. in study done by TTS Paary. Et.al [80] (2016)42% patients had hypertension, 42% cases were diabetic, 25% cases had chronic kidney disease[80]. In study done by Jalal HKT et. al. 51.3% cases were diabetic, 62% hypertensive, 26.6% with chronic kidney disease, 24.3% had coronoary artery disease, 14.7% had CVA. In study done by N.E. HAMMOND et. al. [90] (2022) 45% cases were diabetic, 14.4% with chronic kidney ds. ,9.9% having coronary artery disease, 6% cases had chronic liver disease, 9.9% had h/o CVA, 11.5% had previous respiratory illness[90].

Other medical history

In our study we had post covid-19 ds. patients 5/130 (3.8%), patients having depressive illness 3/130(2.3%), parkinsons, PLHIV, Post RSA- Head injury, Rheumatoid Arthritis, old pulmonary tuberculosis each having 2/130 (1.5%) cases and Tubercular pleural effusion, Rheumatic Heart disease , Burns , B-cell lymphoma, bedridden state, Bipolar disorder, Bullous pemphigoid, Dementia, Carcinoma prostate, extra pulmonary tuberculosis, HCV reactive status, hyperthyroidism, intellectual disability, kypho-scoliosis, motor

neuron disease, P-ANCA vasculitis, Post RSA- abdominal injury, scleroderma, siezure disorder each having 1/130 (0.8%) cases.

PERSONAL HISTORY

In this study 33/130 (25.4%) patients were Alcoholics, 21/130 (16.2%) were smoker and 7/130 (5.4%) were substance abusers.

PRESENTING VITALS

GCS-In this study maximum score was 15 and minimum score of 3 with mean of 11.82. 73/130 (56.2%) patients had GCS in range 13-15 and 27/130 (20.8%) patients had GCS <7. Similar to this study GCS of patients with sepsis was low (9.3%) in study conducted by Jalal HKT et al. [81]

Source of infection

In this study most common source of infection was respiratory tract in 76/130 (58.5%) patients followed by urinary tract 60/130 (46.2%) patients, 25/130 (19.2%) had blood stream infection, 14/130 (10.8%) had skin infection, 12/130 (9.2%) had GIT infection, CNS infection and Genital infection was present in 4/130 (3.1%) patients each, which was similar to study conducted by TS Paary et al. [80](2016)

Similarly in a study conducted by Jalal HKT et al.[81] (2020) respiratory tract infection was present in 46.3 % patients, urinary tract infection in 23.5% patients, cellulitis in 17.9% patients and abdominal sepsis in 12.3% patients.

In this study sputum culture growth was positive in 33/57 (57.9%) patients, urine culture growth was positive in 33/100 (33.0%) patients, pus/wound swab culture was positive in 8/14 (57.1%) patients and blood culture was positive in 24/92 (26.1%) patients, which was comparable to study conducted by Chandrakant M Raibhoge et al.[87] [2019]

Candida (12)% followed by Klebsiella in 6.0%, E. coli and Pseudomonas in 5.0% were the most common pathogens among UTI patients. Predominant pathogens in patients with bloodstream infection were Klebsiella (8.7%), E. coli (5.4%) and Staph. Epidermidis(4.3%). From sputum most common organisms isolated were Acenetobacter (33.3%) and Klebsiella (31.6%).

Among patients with skin and soft tissue infections Klebsiella (35.7%) and Acenetobacter (21.4%) were the most common organisms.

Similar to this study, culture positivity was seen in study by M. Mukhyaprana Prabhu et al.[78] (2012) and Chandrakant M Raibhoge et al.[87] [2019]

Viral Markers

Viral markers positivity was not associated with increased complications of sepsis and mortality. All the patients were not tested for viral markers. Only 8 patients had positive viral markers. This was statistically non-significant.

2D Echocardiography findings

a) **Ventricular Global Hypokinesia (GHK)/Regional wall motion Abnormlaity (RWMA)**

In this study on 2D Echocardiography 84/130 (64.6%) patients had GHK and 46/130 (35.4%) had RWMA which is similar to study by Sua Kim et al.[82](2019).

b) **Ejection Fraction**

Ejection fraction of 20-40 % was in maximum no. of cases 96/130 (73.8%) and ejection fraction of <20 % was in 8/130 (6.2%) cases. Ejection Fraction Of >40 % was in 26 cases (20%). Similar to this study ejection fraction of < 50% was noted in 27.3% cases in study done by M Mukhyaprana Prabhu et al.[78]

SOFA Score

SOFA score was an important predictor of sepsis and sepsis related complications. Patients with increased SOFA were at increased risk of complications. Total of 73/130 (56.15%) cases were having SOFA score between 17-24 and they were at increased risk of mortality with p=0.001 which was statistically significant. Similar to our study conducted by Jalal HKT et al.[81] (2020) 16.3% patients were having SOFA score >15 and 24.5% had SOFA score between 11-15.

Outcome and mortality

35/44(79.5%) Patients who took DAMA got expired as compared to 11/66 (16.7%) patients who were discharged which was statistically significant. (p=0.001).

Mortality status after 30 days of discharge

Due to complications of sepsis 50.8% of patients expired at 30 days after discharge and 49.2% were alive. This was statistically non-significant. Similar rate was seen in study done by Chatterjee et al.(2017)[24].

SUMMARY AND CONCLUSION

A prospective single center observational study of clinical profile and outcome of patients with sepsis and left ventricular systolic dysfunction was done in the medical ICU/HDU of Dayanand Medical College and Hospital, Ludhiana from March 1, 2021 to March 1, 2022.

After meeting the inclusion and exclusion criteria patients were enrolled for study after taking the written informed consent.

- The present study was conducted on 130 patients admitted in medical ICU/HDU with sepsis and LVEF <50%.
- In our study there was a slight male preponderance (85 males and 45 females) for sepsis development with male to female ratio 1.88:1.
- It was concluded that age of patient (p=0.369) and gender of patient (p=0.670) had no statistically significant relation with sepsis development and mortality of patients.
- Patients with sepsis presented with various symptoms. Decreased urine output (p=0.028) and altered mental status/seizures (p=0.019) had statistical significant relationship with sepsis and mortality. Other major presenting symptoms in decreasing order of frequency were fever, shortness of breath, cough, burning micturition, pain abdomen, loose stools and vomiting.
- It was concluded that vitals of patients presented with sepsis were also important predictors of outcome and mortality. Systolic blood pressure and SPO2 were statistically significant for sepsis and mortality with p=0.018 and 0.029 respectively.
- It was concluded that from the co-morbid conditions chronic kidney disease (p=0.027) had statistically significant relationship with sepsis and mortality. Other comorbid conditions were diabetes mellitus, hypertension, COPD, bronchial asthma, CAD,

hypothyroidism, CVA, CLD, these were statistically non-significant.

- Personal history of alcohol consumption and smoking was statistically non-significant for association with sepsis and mortality.
- It was found that from the laboratory investigations low hemoglobin (0.019), increased blood urea (0.002), serum creatinine (0.013) were statistically significant for sepsis and mortality.
- It was concluded that main source of infection in sepsis patients were respiratory tract (76/130) and urinary tract (60/130). Other sources of infection were blood, GIT, skin, CNS and genitalia. All of these were statistically non-significant with association with mortality.
- It was concluded that patients with GCS <7 were more prone to sepsis, sepsis related complications and mortality. This was statistically significant (p=0.039).
- It was concluded that SOFA score was important predictor of sepsis and sepsis induced mortality. Patients with high SOFA score were at increased risk of sepsis and sepsis related complications including mortality. 49/73 patients with SOFA score between17-24 expired with complications of sepsis. This was statistically significant (p=0.001).
- Left ventricular ejection fraction was statistically significant predictor of sepsis related complication and mortality (p=0.535). 50/96 patients admitted with sepsis and LVEF between 20-40 % expired.
- The patients who took DAMA (35/44) from hospital expired more than patients who were discharged (11/66) from the hospital

during 30 day follow up. This was statistically significant with p=0.001).

Conclusion

Hence we concluded that in patients presenting with sepsis and LVEF <50% vital signs including blood pressure and low SPO2 associated with progression of sepsis and mortality due to sepsis. Co-morbid conditions like chronic kidney disease, diabetes mellitus are significantly associated with sepsis development. Respiratory tract and urinary tract are major sources of sepsis development. Low Glasgow coma score at presentation, High SOFA score at presentation and decreased LVEF significantly associated with sepsis and mortality associated with it. Early recognition and Early treatment of sepsis can prevent sepsis related complications and mortality.

BIBLIOGRAPHY

1. Majno G. The ancient riddle of sigma eta psi iota sigma (sepsis). J Infect Dis. 1991;163:937-45.
2. Singer M, Deutschman CS, Seymour CW, Shankar-Hari M, Annane D, Bauer M, et al. The third international consensus defnitions for sepsis and septic shock (Sepsis-3). JAMA. 2016;315:801–10
3. Shankar-Hari M, Phillips GS, Levy ML, Seymour CW, Liu VX, Deutschman CS, et al. Developing a New Definition and Assessing New Clinical Criteria for Septic Shock: For the Third International Consensus Definitions for Sepsis and Septic Shock (Sepsis-3). JAMA. 2016;315:775-87.
4. Sartelli M, Kluger Y, Ansaloni L, Hardcastle TC, Rello J, Watkins RR, et al. Raising concerns about the Sepsis-3 defnitions. World J Emerg Surg. 2018;25:6.
5. Cavaillon JM, Singer M, Skirecki T. Sepsis therapies: learning from 30 years of failure of translational research to propose new leads. EMBO Mol Med. 2020;12:e10128.
6. Cao C, Yu M, Chai Y. Pathological alteration and therapeutic implications of sepsis-induced immune cell apoptosis. Cell Death Dis. 2019;10:782
7. Teggert A, Datta H, Ali Z. Biomarkers for point-of-care diagnosis of sepsis. Micromachines (Basel). 2020;11:286.
8. Choi H, Kim Y, Mirzaaghasi A, Heo J, Kim YN, Shin JH, et al. Exosomebased delivery of super-repressor lκBα relieves sepsis-associated organ damage and mortality. Sci Adv. 2020;6:eaaz6980
9. Fernando SM, Tran A, Taljaard M, Cheng W, Rochwerg B, Seely AJE, et al. Prognostic Accuracy of the Quick Sequential Organ Failure Assessment for Mortality in Patients With Suspected Infection: A Systematic Review and Meta-analysis. Ann Intern Med. 2018;168:266-75.

10. Herwanto V, Shetty A, Nalos M, Chakraborty M, McLean A, Eslick GD, Tang B. Accuracy of Quick Sequential Organ Failure Assessment Score to Predict Sepsis Mortality in 121 Studies Including 1,716,017 Individuals: A Systematic Review and Meta-Analysis. Crit Care Explor. 2019;1:e0043..
11. Serafim R, Gomes JA, Salluh J, Póvoa P. A Comparison of the Quick-SOFA and Systemic Inflammatory Response Syndrome Criteria for the Diagnosis of Sepsis and Prediction of Mortality: A Systematic Review and Meta-Analysis. Chest. 2018;153:646-55.
12. Cinel I, Kasapoglu US, Gul F, Dellinger RP. The initial resuscitation of septic shock. J Crit Care. 2020;57:108-17.
13. Delano MJ, Ward PA. Sepsis-induced immune dysfunction: can immune therapies reduce mortality? J Clin Invest. 2016;126:23–31.
14. Fleischmann C, Scherag A, Adhikari NK, Hartog CS, Tsaganos T, Schlattmann P, et al. Assessment of Global Incidence and Mortality of Hospital-treated Sepsis. Current Estimates and Limitations. Am J Respir Crit Care Med. 2016;193:259-72.
15. Jawad I, Lukšić I, Rafnsson SB. Assessing available information on the burden of sepsis: global estimates of incidence, prevalence and mortality. J Glob Health. 2012;2:010404.
16. Becker JU, Theodosis C, Jacob ST, Wira CR, Groce NE. Surviving sepsis in low-income and middle-income countries: new directions for care and research. Lancet Infect Dis. 2009;9:577-82.
17. Rudd KE, Johnson SC, Agesa KM, Shackelford KA, Tsoi D, Kievlan DR, et al. Global, regional, and national sepsis incidence and mortality, 1990-2017: analysis for the Global Burden of Disease Study. Lancet. 2020 Jan 18;395:200-211.
18. CDC. Sepsis. https://www.cdc.gov/sepsis/index.html. Accessed on 23-02-2023.
19. Schultz MJ, Dunser MW, Dondorp AM, Adhikari NKJ, Iyer S, Kwizera A, et al. Current challenges in the management of sepsis in ICUs in

resource-poor settings and suggestions for the future. Intensive Care Med. 2017;43:612–24.

20. GBD 2016 Healthcard Access and Quality Collaborators. Measuring performance on the Healthcare Access and Quality Index for 195 countries and territories and selected subnational locations: a systematic analysis from the Global Burden of Disease Study 2016. Lancet. 2018;391:2236-71.
21. Reinhart K, Daniels R, Kissoon N, Machado FR, Schachter RD, Finfer S. Recognizing sepsis as a Global Healthpriority–a WHO resolution.N Engl JMed 2017;377:414–7
22. Fleischmann-Struzek C, Mellhammar L, Rose N, Cassini A, Rudd KE, Schlattmann P, et al. Incidence and mortality of hospital- and ICU-treated sepsis: results from an updated and expanded systematic review and meta-analysis. Intensive Care Med. 2020;46:1552-62.
23. Dugani S, Veillard J, Kissoon N. Reducing the global burden of sepsis. CMAJ. 2017;189:E2-E3.
24. Todi, S, Chatterjee, S, Bhattacharyya M. Epidemiology of Severe Sepsis in India. *Crit. Care* **2007**;*11*:65.
25. Mayr, F.B, Yende, S, Angus D.C. Epidemiology of Severe Sepsis. *Virulence* **2014**;*5*:4–11.
26. Sakr Y, Jaschinski U, Wittebole X, et al. Sepsis in Intensive Care Unit Patients: Worldwide Data From the Intensive Care over Nations Audit. Open Forum Infect Dis. 2018;5:ofy313.
27. Ma X, Liu B, Fan L, Liu Y, Zhao Y, Ren T, et al. Native and engineered exosomes for inflammatory disease. Nano Res. 2022:1-16.
28. Hotchkiss RS, Monneret G, Payen D. Sepsis-induced immunosuppression: from cellular dysfunctions to immunotherapy. Nat Rev Immunol. 2013;13:862–74.
29. Needham DM, Davidson J, Cohen H, Hopkins RO, Weinert C, Wunsch H, et al. Improving long-term outcomes after discharge from intensive

care unit: report from a stakeholders' conference. Crit Care Med. 2012;40:502–9.

30. Liu D, Huang SY, Sun JH, Zhang HC, Cai QL, Gao C, et al. Sepsis-induced immunosuppression: mechanisms, diagnosis and current treatment options. Mil Med Res. 2022;9:56.
31. Evans T. Diagnosis and management of sepsis. Clin Med. 2018;18:146–9.
32. Deutschman CS, Tracey KJ. Sepsis: current dogma and new perspectives. Immunity. 2014;40:463–75.
33. Geri G, Vignon P, Aubry A, Fedou AL, Charron C, Silva S, et al. Cardiovascular clusters in septic shock combining clinical and echocardiographic parameters: a post hoc analysis. *Intensive Care Med.* 2019;45:657–67.
34. Akira S, Uematsu S, Takeuchi O. Pathogen recognition and innate immunity. Cell. 2006;124:783–801.
35. Russell JA, Boyd J, Nakada T, Thair S, Walley KR. Molecular mechanisms of sepsis. Contrib Microbiol. 2011;17:48–85
36. Bianchi ME. DAMPs, PAMPs and alarmins: all we need to know about danger. J Leukoc Biol. 2007;81:1–5
37. Adib-Conquy M, Cavaillon JM. Host inflammatory and anti-inflammatory response during sepsis. Pathol Biol (Paris). 2012;60:306–13
38. Soriano FG, Lorigados CB, Pacher P, Szabó C. Effects of a potent peroxynitrite decomposition catalyst in murine models of endotoxemia and sepsis. Shock. 2011;35:560–6
39. Torres-Dueñas D, Celes MR, Freitas A, Alves-Filho JC, Spiller F, Dal-Secco D, et al. Peroxynitrite mediates the failure of neutrophil migration in severe polymicrobial sepsis in mice. Br J Pharmacol. 2007;152:341–52
40. Vieillard-Baron A. Septic cardiomyopathy. Ann Intensive Care. 2011;1:6.

41. Poelaert J, Declerck C, Vogelaers D, Colardyn F, Visser CA. Left ventricular systolic and diastolic function in septic shock. Intensive Care Med. 1997 May;23:553-60.
42. Jardin F, Fourme T, Page B, Loubières Y, Vieillard-Baron A, Beauchet A, et al. Persistent preload defect in severe sepsis despite fluid loading: A longitudinal echocardiographic study in patients with septic shock. Chest. 1999;116:1354-9.
43. Gyawali B, Ramakrishna K, Dhamoon AS. Sepsis: The evolution in definition, pathophysiology, and management. SAGE Open Med. 2019;7:2050312119835043
44. Vincent J-L. The clinical challenge of sepsis identifcation and monitoring. PLoS Med. 2016;13:e1002022
45. Ifedayo Kuye CR. Spotlight: Overdiagnosis and Delay: Challenges in Sepsis Diagnosis https://psnet.ahrq.gov/web-mm/spotlight-overdiagnosis-and-delay-challenges-sepsis-diagnosis. Accessed 23-02-2023
46. Hotchkiss RS, Moldawer LL, Opal SM, Reinhart K, Turnbull IR, Vincent J-L. Sepsis and septic shock. Nat Rev Dis Prim. 2016;2:16045
47. Kumar S, Tripathy S, Jyoti A, Singh SG. Recent advances in biosensors for diagnosis and detection of sepsis: a comprehensive review. Biosens Bioelectron. 2019;124–125:205–15.
48. Carrigan SD, Scott G, Tabrizian M. Toward resolving the challenges of sepsis diagnosis. Clin Chem. 2004;50:1301–14
49. Samraj RS, Zingarelli B, Wong HR. Role of biomarkers in sepsis care. Shock. 2013;40:358–65
50. Henriquez-Camacho C, Losa J. Biomarkers for Sepsis. Biomed Res Int. 2014;2014:547818
51. Van Engelen TSR, Wiersinga WJ, Scicluna BP, van der Poll T. Biomarkers in sepsis. Crit Care Clin. 2018;34:139–52

52. Póvoa P, Coelho L, Almeida E, Fernandes A, Mealha R, Moreira P, et al. C-reactive protein as a marker of infection in critically ill patients. Clin Microbiol Infect. 2005;11:101–8
53. Gunsolus IL, Sweeney TE, Liesenfeld O, Ledeboer NA. Diagnosing and Managing Sepsis by Probing the Host Response to Infection: Advances, Opportunities, and Challenges. J Clin Microbiol. 2019;57:e00425-19
54. Wacker C, Prkno A, Brunkhorst FM, Schlattmann P. Procalcitonin as a diagnostic marker for sepsis: a systematic review and meta-analysis. Lancet Infect Dis. 2013;13:426-35.
55. Alhazzani W, Møller MH, Arabi YM, Loeb M, Gong MN, Fan E, et al. Surviving Sepsis Campaign: Guidelines on the Management of Critically Ill Adults with Coronavirus Disease 2019 (COVID-19). Crit Care Med. 2020;48:e440-69.
56. Molnár Z, Giamarellos-Bourboulis EJ, Kumar A, Nierhaus A. Sepsis: diagnostic and therapeutic challenges. Biomed Res Int. 2016;2016:5786182
57. Mehrunnissa Taj, Michael Brenner, Zeenat Sulaiman, Vinciya Pandian. Sepsis protocols to reduce mortality in resource-restricted settings: A systematic review,Intensive and Critical Care Nursing.2022;72:1032.
58. Kumar A, Roberts D, Wood KE, Light B,Parrillo JE, Sharma S et al. Duration ofhypotension before initiation ofeffective antimicrobial therapy is thecritical determinant of survival inhuman septic shock.Crit Care Med 2006;34:1589–96.
59. Seymour CW, Gesten F, Prescott HC, Friedrich ME, Iwashyna TJ, Phillips GS et al. Time to treatment and mortalityduring mandated emergency Care forSepsis.NEnglJMed 2017;376:2235–44.
60. Shorr AF, Micek ST, Welch EC, Doherty JA, Reichley RM, Kollef MH. Inappropriate antibiotic therapy in Gram-negative sepsis increases hospital length of stay. Crit Care Med. 2011;39:46-51.

61. Cheah AL, Spelman T, Liew D, Peel T, Howden BP, Spelman D, Grayson ML, Nation RL, Kong DC. Enterococcal bacteraemia: factors influencing mortality, length of stay and costs of hospitalization. Clin Microbiol Infect. 2013;19:E181-9.
62. Parker MM, Shelhamer JH, Bacharach SL, Green MV, Natanson C, Frederick TM et al. Profound but reversible myocardial depression in patients with septic shock. Ann InternMed1984;100: 483–90.
63. Marik PE, Farkas JD. The changing paradigm of sepsis: early diagnosis, early antibiotics, early pressors, and early adjuvant treatment. Crit Care Med. 2018;46:1690–2.
64. De Backer D, Cecconi M, Lipman J, Machado F, Myatra SN, Ostermann M, et al. Challenges in the management of septic shock: a narrative review. Intensive Care Med. 2019;45:420–33.
65. Rhodes A, Evans LE, Alhazzani W, Levy MM, Antonelli M, Ferrer R, et al. Surviving sepsis campaign: international guidelines for management of sepsis and septic shock: 2016. Intensive Care Med. 2017;43:304–77.
66. Surviving Sepsis Campaign Bundles.[cited 2018 Dec 28]. Available from URL: http://www.survivingsepsis.org/Bundles/Pages/default.aspx
67. Annane D, Bellissant E, Bollaert PE, Briegel J, Confalonieri M, De Gaudio R et al. Corticosteroids in the treatment ofsevere sepsis and septic shock in adults:a systematic review.JAMA 2009;301:2362–75.
68. Venkatesh B, Finfer S, Cohen J, Rajbhandari D, Arabi Y, Bellomo R et al.Adjunctive glucocorticoid therapy inpatients with septic shock.N Engl J Med2018;378:797–808
69. Annane D, Renault A, Brun-Buisson C, Megarbane B, Quenot JP, Siami S et al. Hydrocortisone plusfludrocortisone foradults with septic shock. N Engl J Med2018;378:809–18
70. Venkatesh B, Finfer S, Myburgh J,Cohen J, Billot L. Long-term outcomesof the ADRENAL trial.N Engl J Med 2018;378:1744–5

71. Garnacho-Montero J, Garcia-Garmendia JL, Barrero-Almodovar A, Jimenez-Jimenez FJ, Perez-Paredes C, Ortiz-Leyba C. Impact of adequate empirical antibiotic therapy on the outcome of patients admitted to the intensive care unit with sepsis*. Crit Care Med. 2003;31:2742–51
72. Busani S, Roat E, Serafni G, Mantovani E, Biagioni E, Girardis M. The role of adjunctive therapies in septic shock by gram negative MDR/XDR infections. Can J Infect Dis Med Microbiol. 2017;2017:2808203
73. Uppu DSSM, Ghosh C, Haldar J. Surviving sepsis in the era of antibiotic resistance: are there any alternative approaches to antibiotic therapy? Microb Pathog. 2015;80:7–13.
74. Charlton M, Thompson JP. Pharmacokinetics in sepsis. BJA Educ. 2019;19:7–13
75. Selvaraj V, Manne ND, Arvapalli R, Rice KM, Nandyala G, Fankenhanel E, et al. Efect of cerium oxide nanoparticles on sepsis induced mortality and NF-κB signaling in cultured macrophages. Nanomedicine. 2015;10(8):1275–88.
76. Ouellette DR, Shah SZ. Comparison of outcomes from sepsis between patients with and without pre-existing left ventricular dysfunction: a case-control analysis. *Crit Care*. 2014;18:R79.
77. Finfer S, Bellomo R, Lipman J, French C, Dobb G, Myburgh J. Adult-population incidence of severe sepsis in Australian and New Zealand Intensive Care Units. Intensive Care Med. 2004;30:589–96.
78. Prabhu MM, Yalakala SK, Shetty R, Thakkar A, Sitapara T. Prognosis of left ventricular systolic dysfunction in septic shock patients. J Clin Diagn Res. 2015 ;9: OC05-8.
79. Pulido JN, Afessa B, Masaki M, Yuasa T, Gillespie S, Herasevich V, et al. Clinical spectrum, frequency, and significance of myocardial dysfunction in severe sepsis and septic shock. Mayo Clin Proc. 2012;87:620-8

80. Paary T T, Kalaiselvan M S, Renuka M K, Arunkumar A S. Clinical profile and outcome of patients with severe sepsis treated in an intensive care unit in India. Ceylon Med J. 2016 Dec 30;61(4):181-84.
81. Tharappel Jalal H.K, Thomas T. P., George A.A, & Mohammed H. Epidemiology of sepsis and its various characteristics in a tertiary care adult-multidisciplinary ICU in South India: a retrospective study. *International Journal of Research in Medical Sciences*, 2020;*8*:1260–65.
82. Kim S, Lee JD, Kim BK, Kim YH, Kim JH. Association between Left Ventricular Systolic Dysfunction and Mortality in Patients with Septic Shock. J Korean Med Sci. 2020;35:e24.
83. Abou Dagher G, Hajjar K, Khoury C, El Hajj N, Kanso M, Makki M, Mailhac A, Bou Chebl R. Outcomes of patients with systolic heart failure presenting with sepsis to the emergency department of a tertiary hospital: a retrospective chart review study from Lebanon. BMJ Open. 2018;8:e022185.
84. Vallabhajosyula S, Jentzer JC, Geske JB, Kumar M, Sakhuja A, Singhal A, et al. New-Onset Heart Failure and Mortality in Hospital Survivors of Sepsis-Related Left Ventricular Dysfunction. Shock. 2018;49:144-49.
85. Cirulis MM, Huston JH, Sardar P, Suksaranjit P, Wilson BD, Hatton ND, et al. Right-to-left ventricular end diastolic diameter ratio in severe sepsis and septic shock. J Crit Care. 2018 Dec;48:307-310.
86. Walker AMN, Drozd M, Hall M, Patel PA, Paton M, Lowry J, et al. Prevalence and Predictors of Sepsis Death in Patients With Chronic Heart Failure and Reduced Left Ventricular Ejection Fraction. J Am Heart Assoc. 2018;7: e009684.
87. Chandrakant M Raibhoge, Kiran Mali. A study of clinical profile of sepsis in patients admitted in Intensive care unit at tertiary care hospital. MedPulse International Journal of Medicine. March 2022;21:78-83.

88. Shin DG, Kang MK, Seo YB, Choi J, Choi SY, Choi Set al. Factors associated with abnormal left ventricular ejection fraction (decreased or increased) in patients with sepsis in the intensive care unit. PLoS One. 2020;15: e0229563.
89. Liu VX, Bhimarao M, Greene JD, Manickam RN, Martinez A, Schuler A, et al. The Presentation, Pace, and Profile of Infection and Sepsis Patients Hospitalized Through the Emergency Department: An Exploratory Analysis. Crit Care Explor. 2021 Feb 24;3(3):e0344.
90. Hammond NE, Kumar A, Kaur P, Tirupakuzhi Vijayaraghavan BK, Ghosh A, et al. Estimates of Sepsis Prevalence and Outcomes in Adult Patients in the ICU in India: A Cross-sectional Study. Chest. 2022;161:1543-54.
91. Muehlberg F., Blaszczyk, E., Will, K., Wilczek, S., Brederlau, J., and Schulz-Menger J. Characterization of critically ill patients with septic shock and sepsis-associated cardiomyopathy using cardiovascular MRI. ESC Heart Failure, 2022;9:2147–56.
92. Dugar S, Sato R, Chawla S, Young J, Wang X, Grimm R, Collier P, Lanspa M, Duggal A. Is Left Ventricular Systolic Dysfunction Associated With Increased Mortality Among Patients With Sepsis and Septic Shock? Chest. 2023:S0012-3692;00111-3.
93. Simon L, Gauvin F, Amre DK, Saint-Louis P, Lacroix J. Serum procalcitonin and C-reactive protein levels as markers of bacterial infection: a systematic review and meta-analysis. Clin Infect Dis. 2004;39:206-17.
94. Engel C, Brunkhorst FM, Bone HG, Brunkhorst R, Gerlach H, Grond S, et al. Epidemiology of sepsis in Germany: results from a national prospective multicenter study. Intensive Care Med. 2007;33:606-18.

S. No	MRD	INITIALS	AGE	AGE GROUP	SEX	DOA	DOD	FEVER	COUGH/ SPUTUM	PAIN ABDOMEN	DECREASED URINE OUTPUT	BURNING/ INCEASED FREQUENCY OF URINATION	LOOSE STOOLS	ALTRED SENSORIUM/ SIEZURES	SOB	VOMITING/ NAUSEA	OTHERS	DM	HTN	COPD/B.ASTHMA/ANY RESP DISORDER	CLD
1	1308834	SS	62	5	M	22-05-2021	12-06-2021	Y	N	N	N	N	N	N	N	N	N	N	N	N	N
2	1314039	NK	63	5	F	30-05-2021	12-06-2021	Y	N	N	N	N	Y	Y	N	Y	N	N	N	N	N
3	999518	AS	45	3	M	04-06-2021	08-06-2021	N	N	N	N	N	N	N	Y	N	N	N	Y	N	N
4	1315468	SS	38	2	M	01-06-2021	23-06-2021	N	Y	N	N	N	N	N	Y	N	N	N	N	Y	N
5	1198413	SP	70	5	M	31-05-2021	03-06-2021	N	N	N	Y	N	N	N	N	N	BLOOD IN VOMITUS	Y	Y	N	N
6	945977	KS	56	4	M	28-05-2021	15-06-2021	N	Y	N	N	N	N	Y	N	N	N	Y	N	N	N
7	1312440	BJS	62	5	M	27-05-2021	11-07-2021	N	N	N	N	N	N	N	Y	N	PEDAL SWELLING	Y	Y	N	N
8	1308883	NA	62	5	F	23-05-2021	09-06-2021	Y	N	N	N	N	N	N	N	N	LETHARGY	N	N	N	N
9	1308751	PS	78	6	M	22-05-2021	16-06-2021	Y	Y	N	N	N	N	N	Y	N	LETHARGY	Y	N	N	N
10	688165	KL	78	6	M	04-06-2021	14-06-2021	N	N	N	Y	N	N	Y	N	N	N	Y	Y	N	N
11	1316788	GS	20	1	M	03-06-2021	09-06-2021	N	N	N	N	N	N	N	N	N	OPC POISONING	N	N	N	N
12	1318291	UR	70	5	F	06-06-2021	11-06-2021	N	N	N	N	N	N	N	Y	N	N	N	Y	N	N
13	1312474	HS	28	1	M	27-05-2021	23-06-2021	Y	N	Y	Y	N	N	N	N	N	N	N	N	N	N
14	1317402	KS	65	5	M	04-06-2021	20-06-2021	N	Y	N	Y	N	N	Y	Y	N	N	N	Y	Y	N
15	1313135	BR	55	4	M	05-06-2021	29-06-2021	N	Y	N	N	N	N	N	Y	N	N	Y	Y	N	N
16	1317416	GK	34	2	F	04-06-2021	12-06-2021	N	N	N	Y	N	N	N	Y	N	N	N	N	N	N
17	1313289	SC	86	6	M	29-05-2021	07-06-2021	Y	N	N	N	N	N	N	N	N	LOSS OF APPETITE	Y	Y	N	N
18	1313327	SK	58	4	F	30-05-2021	04-06-2021	N	N	Y	N	N	N	Y	Y	N	PEDAL SWELLING	Y	Y	N	N
19	1311126	JD	36	2	F	26-05-2021	05-06-2021	Y	N	N	N	N	N	N	Y	N	N	Y	Y	N	N
20	1229583	B	50	3	F	03-06-2021	07-06-2021	N	Y	N	Y	N	N	Y	Y	N	BODY SWELLING	Y	Y	N	N
21	1313107	AK	48	3	M	28-05-2021	03-06-2021	Y	N	N	Y	Y	N	Y	N	N	N	Y	N	N	N
22	1312496	VKK	76	6	M	27-05-2021	08-06-2021	Y	Y	N	N	N	N	N	Y	N	N	Y	Y	Y	N
23	1119943	HS	65	5	M	08-06-2021	22-06-2021	N	Y	N	N	N	N	N	N	N	LOSS OF APPETITE	Y	Y	N	N
24	1134066	RS	66	5	M	09-07-2021	18-07-2021	Y	N	N	N	N	Y	Y	N	N	N	N	Y	N	N
25	1236486	KS	25	1	M	09-01-2022	21-01-2022	Y	Y	N	N	N	N	N	Y	N	N	N	Y	N	N
26	1234438	SS	50	3	M	05-01-2022	21-01-2022	N	N	N	N	N	N	N	Y	N	SKIN TIGHTENING	N	N	N	N
27	1204887	JK	60	4	F	09-03-2021	14-03-2021	N	N	N	N	N	N	N	Y	N	N	Y	N	N	N
28	1266238	DD	63	5	F	08-03-2021	13-03-2021	Y	Y	N	N	N	N	N	N	N	N	N	N	N	N
29	1265486	MS	21	1	M	07-03-2021	22-03-2021	Y	N	N	N	N	Y	N	Y	N	JAUNDICE	N	N	N	N
30	1131733	VKL	60	4	M	17-01-2022	02-02-2022	N	N	N	N	N	N	Y	N	N	N	Y	Y	N	N
31	966910	NS	63	5	M	21-02-2022	24-01-2022	Y	N	N	Y	N	N	N	N	N	DIZZINESS	Y	Y	N	Y
32	445706	ALG	63	5	M	13-01-2022	23-01-2022	Y	Y	N	N	N	N	N	Y	N	BLOOD IN SPUTUM	N	N	N	N
33	1210090	PA	46	3	M	18-11-2021	18-12-2021	Y	Y	N	N	N	N	N	Y	N	N	Y	N	N	N
34	1118983	JK	65	5	F	06-06-2021	23-06-2021	Y	N	N	N	N	N	N	Y	N	N	N	Y	N	N
35	1118810	BS	25	1	M	05-06-2021	22-06-2021	Y	N	N	N	N	N	N	N	N	BLACK STOOLS	N	N	N	N
36	1118864	SK	28	1	M	06-06-2021	16-06-2021	N	N	N	Y	N	N	N	N	Y	SNAKE BITE	N	N	N	N
37	492838	GK	68	5	M	02-06-2021	25-06-2021	Y	Y	N	N	N	N	N	N	N	FACIAL SWELLING	Y	Y	N	N
38	1122943	JK	59	4	F	07-07-2021	15-07-2021	N	N	N	Y	N	N	Y	N	N	N	Y	Y	N	N
39	45946	SK	55	4	M	15-09-2021	23-09-2021	N	N	N	N	N	N	N	N	N	RIGHT HEMIPARESIS	Y	N	N	N
40	1173418	DS	62	5	M	14-09-2021	24-09-2021	Y	N	Y	N	N	N	Y	Y	N	N	N	N	N	N
41	1171335	ML	63	5	F	11-09-2021	04-10-2021	N	N	Y	N	N	N	N	Y	N	ABDOMEN DISTENSION	N	N	N	N
42	1113188	SS	52	4	F	03-09-2021	21-09-2021	Y	N	N	Y	N	N	Y	Y	N	SLURRED SPEECH	Y	N	N	N
43	891124	JL	71	6	F	08-09-2021	13-09-2021	Y	N	N	N	N	N	N	Y	N	N	Y	N	N	N
44	1169277	M	56	4	M	07-09-2021	23-09-2021	N	N	N	N	N	N	Y	N	N	CELPHOS POISON	Y	N	N	N
45	1167714	RR	33	2	F	05-09-2021	08-09-2021	Y	N	N	Y	N	N	N	N	N	N	N	N	N	N
46	1048315	CL	63	5	M	02-09-2021	08-09-2021	N	N	Y	Y	N	N	Y	N	N	N	Y	Y	N	N
47	1164650	SK	50	3	F	31-08-2021	11-09-2021	Y	Y	N	N	N	N	N	Y	N	N	N	Y	Y	N
48	1163484	HP	56	4	M	29-08-2021	06-09-2021	Y	N	N	N	N	N	Y	N	N	LOSS OF APPETITE	Y	N	N	N
49	1163390	PR	55	4	M	29-08-2021	04-09-2021	N	N	N	N	N	N	Y	N	N	LETHARGY	N	N	N	N

S. No	MRD	CKD	CAD	HYPOTHYROIDISM	CVA	OTHER MEDICAL HISTORY	SURGICAL HISTORY	ALCHOL	SMOKING	OTHER SUBSTANCE	GCS	gcs GROUP	SBP	DBP	HR	RR	SPO2%	FIO2 %	RTI	UTI	GIT	SKIN	BLOOD STREAM	CNS-INFECTION
1	1308834	N	N	N	N	N	N	Y	N	OPIOID	9	2	100	60	120	22	95	50	N	N	N	Y	N	N
2	1314039	N	N	N	N	RA	N	N	N	N	8	1	60	40	96	24	88	36	N	Y	N	N	N	N
3	999518	Y	Y	N	N	N	N	N	N	N	9	2	200	100	140	20	50	21	Y	N	N	N	N	N
4	1315468	N	N	N	N	P-ANCA VASCULITIS, RA	N	N	N	N	15	3	120	70	92	35	50	21	N	N	N	N	N	N
5	1198413	N	Y	N	N	N	POST CABG	Y	N	N	13	3	90	60	110	24	92	21	N	Y	N	N	N	N
6	945977	N	N	N	N	POST RSA-HEAD INJURY	N	Y	N	N	4	1	60	40	126	20	99	100	Y	Y	N	N	Y	N
7	1312440	N	Y	N	Y	N	POST CABG	N	N	N	14	3	120	70	90	24	94	100	Y	Y	N	N	N	N
8	1308883	N	N	N	N	POST RSA-ABD. INJURY	POST RSA ABD. Sx	N	N	N	15	3	80	60	90	16	96	21	N	N	N	Y	N	N
9	1308751	N	Y	N	N	EXTRA PULM. TB	N	Y	N	N	15	3	70	40	86	24	86	21	Y	N	Y	N	N	N
10	688165	Y	N	N	N	N	ARTHROPLASTY	N	N	N	12	2	120	90	98	22	97	50	Y	N	N	N	N	N
11	1316788	N	N	N	N	N	N	N	N	N	3	1	140	70	152	12	98	50	Y	N	N	N	N	N
12	1318291	Y	N	N	N	N	N	N	N	N	3	1	100	60	93	18	92	100	Y	N	N	N	N	N
13	1312474	N	N	N	N	N	N	Y	N	N	15	3	120	80	68	22	97	21	N	N	Y	N	Y	N
14	1317402	N	Y	N	N	OLD PULM. TB	N	N	N	N	11	2	120	70	74	26	89	100	Y	Y	N	N	N	N
15	1313135	Y	Y	N	N	N	N	N	N	N	15	3	120	70	130	24	90	100	Y	Y	N	N	N	N
16	1317416	N	N	N	N	N	POST LSCS	N	N	N	15	3	100	60	144	28	88	21	N	Y	N	N	N	N
17	1313289	N	Y	N	N	HCV REACTIVE	N	N	N	N	14	3	140	70	100	24	95	21	Y	Y	N	N	Y	N
18	1313327	N	N	Y	N	N	N	N	N	N	9	2	90	60	100	24	97	100	N	N	Y	N	N	N
19	1311126	N	N	N	N	RA	N	N	N	N	15	3	120	70	126	34	84	45	Y	N	N	N	N	N
20	1229583	Y	Y	N	N	OLD PULM. TB	N	N	N	N	12	2	130	80	80	30	68	21	N	Y	N	N	N	N
21	1313107	N	N	N	N	N	N	N	Y	N	13	3	100	60	90	16	92	60	N	Y	N	N	Y	N
22	1312496	N	N	N	N	BULLOUS PEMPHIGOID	TOTAL KNEE REPLACEMENT	N	Y	N	14	3	150	100	144	28	89	21	Y	Y	N	N	N	N
23	1119943	N	Y	N	N	N	POST PTCA	Y	N	N	15	3	110	70	94	20	92	21	Y	N	N	N	Y	N
24	1134066	N	N	N	Y	N	N	Y	N	N	8	1	80	60	84	16	92	100	Y	N	N	N	Y	N
25	1236486	N	N	N	N	N	N	Y	Y	OPIOID	7	1	130	90	78	16	100	100	Y	N	N	N	N	N
26	1234438	N	N	N	N	SCLERODERMA	N	N	N	N	15	3	180	100	124	22	80	21	Y	N	N	N	N	N
27	1204887	N	Y	N	N	N	N	N	N	N	15	3	80	40	82	22	97	21	Y	N	N	N	N	N
28	1266238	N	Y	N	N	N	POST PTCA	N	N	N	8	1	60	30	116	20	94	100	Y	N	N	N	N	N
29	1265486	N	N	N	N	N	N	N	N	N	15	3	60	30	130	20	96	21	N	Y	N	N	N	N
30	1131733	Y	Y	N	N	N	POST PTCA	N	N	N	3	1	40	20	68	18	100	100	Y	Y	N	N	N	N
31	966910	N	N	N	N	N	N	Y	N	N	15	3	80	50	92	24	98	21	N	Y	N	N	N	N
32	445706	N	N	N	N	POST COVID	N	N	N	N	15	3	60	30	160	22	84	21	Y	N	N	N	N	N
33	1210090	N	N	N	N	N	N	N	N	N	15	3	80	40	86	24	93	100	N	N	N	N	N	N
34	1118983	N	N	N	N	N	N	N	N	N	15	3	170	110	118	28	67	21	N	Y	N	N	N	N
35	1118810	N	N	N	N	N	N	N	N	N	15	3	60	30	100	36	88	50	N	Y	N	N	Y	N
36	1118864	N	N	N	N	EXTRA PULM. TB	N	N	N	N	15	3	130	80	100	22	91	21	N	N	N	Y	N	N
37	492838	N	Y	N	N	N	POST PTCA	Y	N	N	15	3	140	80	110	24	93	21	Y	N	N	N	N	N
38	1122943	Y	Y	Y	N	N	N	N	N	N	13	3	110	70	82	20	98	21	N	Y	Y	Y	Y	N
39	45946	N	N	N	N	N	FOOT AMPUTATION	N	N	N	13	3	110	70	94	18	96	21	N	N	N	Y	N	N
40	1173418	N	N	N	N	INTELLECTUAL DISABILITY	N	N	N	N	13	3	60	30	122	24	64	21	N	N	N	N	N	N
41	1171335	N	N	N	N	N	N	Y	Y	N	13	3	60	30	110	28	72	21	N	N	Y	Y	N	N
42	1113188	Y	N	N	N	N	N	N	N	N	12	2	90	60	99	26	76	21	N	Y	N	N	N	N
43	891124	N	Y	Y	N	N	N	N	N	N	15	3	100	60	122	28	68	21	Y	N	N	N	Y	N
44	1169277	N	N	N	N	N	N	Y	N	OPIOID	9	2	60	30	127	32	84	21	Y	N	N	N	N	N
45	1167714	N	N	N	N	N	POST LSCS	N	N	N	11	2	80	60	122	28	81	21	N	Y	N	N	Y	N
46	1048315	N	Y	N	N	N	POST PTCA	Y	N	N	9	2	60	40	92	28	72	21	N	Y	Y	N	N	N
47	1164650	N	Y	N	N	N	N	N	N	N	9	2	90	60	102	20	68	21	Y	N	N	N	N	N
48	1163484	N	N	N	N	BIPOLAR DISORDER	N	Y	N	N	13	3	110	70	116	28	88	21	N	Y	N	N	N	N
49	1163390	N	N	N	N	B-CELL LYMPHOMA	N	Y	N	N	3	1	90	60	122	16	94	100	Y	N	N	N	N	N

S. No	MRD	GENITAL INFECTION	TLC (X 1000/ UL)	HB (gm/dl)	PLATELETS (X 1000/ UL)	UREA (mg/dl)	CREATININE (mg/dl)	SERUM SODIUM (mmol/L)	S. POTASSIUM (mmol/L)	S. CHLORIDE (mmol/L)	S. CALCIUM	S. MAGNESIUM	S. PHOSPHORUS	TOTAL BILIRUBIN (mg/dl)	SGOT (U/L)	SGPT (U/L)
1	1308834	N	8.7	13.4	150	86	2.42	131	5.06	98	8.6	1.45	4.7	2.56	42	44
2	1314039	N	15.79	8.4	320	40	1.35	133	3.9	104	12	2.49	2.4	0.36	32	10
3	999518	N	14.5	11.3	188	117	7.82	140	7.12	97	10.3	2.81	6.52	0.65	30	78
4	1315468	N	3.3	13.5	150	52	1.28	144	4.26	103	9.9	2.37	3.04	0.93	40	112
5	1198413	N	11.4	9	157	113	3.31	138	5.1	100	-	-	-	1.62	292	73
6	945977	N	40	7.3	110	81	2.21	145	4.1	111	7.8	1.66	2.9	0.69	88	68
7	1312440	N	16	9.7	279	83	3.69	127	4.12	91	8.1	-	2.83	0.44	28	19
8	1308883	N	18.6	11.5	150	41	0.71	139	3.8	103	8.5	2.64	1.29	2.16	53	17
9	1308751	N	22	9.7	203	90	3.33	120	4.84	86	8.5	1.95	1.9	5.68	57	143
10	688165	N	9.5	9.8	193	16.4	8.36	135	5.5	98	8.2	2.2	7.5	0.73	17	17
11	1316788	N	13.8	14.7	262	17	0.9	144	3.27	101	9.7	1.69	3.56	0.57	23	18
12	1318291	N	15.3	6.7	207	261	7.15	143	4.38	104	-	-	-	0.49	23	16
13	1312474	N	9.9	14.2	152	123	6.39	142	3.9	104	8.7	1.97	3.76	2.95	2735	382
14	1317402	N	25	10.1	292	118	2.49	147	5.26	112	8.8	2.23	7.2	0.77	58	32
15	1313135	N	8.7	12.2	245	76	2.17	135	4.8	102	-	-	-	0.6	26	26
16	1317416	N	35.5	10.5	260	20	0.54	141	4.43	109	8.1	1.35	4.65	0.61	56	87
17	1313289	N	17.5	9.4	60	237	3.28	129	5.49	100	8.2	2.67	6.7	0.66	22	33
18	1313327	N	9.3	14.2	156	56	1.13	146	5.01	91	8.2	1.56	1.4	1.39	75	23
19	1311126	N	26	8.1	481	26	0.57	135	3.7	85	8	1.75	3.29	0.31	30	8
20	1229583	N	8.8	7.6	393	89	2.74	132	5.3	101	7.7	2.37	7.42	0.12	22	11
21	1313107	N	11.8	5.9	60	203	5.2	137	4.7	98	9.3	2.6	4.48	0.32	11	4
22	1312496	N	15.6	13.7	141	48	1.84	136	3.5	97	10	2.32	3.9	0.53	162	31
23	1119943	N	15.7	11.6	267	59	1.18	124	3.33	92	7.2	2.96	2.4	0.58	53	62
24	1134066	N	18	13.7	28	102	2.87	138	3.6	99	7.7	1.87	1.7	1.42	651	212
25	1236486	N	17.6	13	216	47	0.88	150	3.23	111	7.5	2.13	1.9	1.3	49	516
26	1234438	N	13.5	11.4	100	117	3.78	138	4.32	102	-	-	-	0.86	20	17
27	1204887	N	13.6	12.8	266	152	1.6	125	5.02	84	8.4	-	-	1.95	167	156
28	1266238	N	24.5	13.7	154	46	1.21	145	4.6	112	7.9	2.03	1.73	0.24	64	30
29	1265486	N	19.2	13.4	54	74	4.91	141	4.9	102	-	-	-	7.71	38	37
30	1131733	N	10.4	3.9	216	213	5.9	136	4.72	97	-	-	-	0.77	12	6
31	966910	N	9.9	9.2	110	93	3.29	136	5.17	103	8.9	1.96	2.54	0.52	20	23
32	445706	N	18	11.6	264	58	2.03	140	3.18	99	8.6	1.88	1.87	1.67	29	28
33	1210090	N	18	15.3	156	58	0.74	144	4.9	110	8.1	2.31	2.32	0.75	53	80
34	1118983	N	16.6	9.4	140	266	4.64	132	4.08	93	8.6	2.31	8.11	0.54	35	13
35	1118810	N	40.6	12.4	100	258	6.34	139	6.63	100	6.5	3.08	14	2.9	2681	1696
36	1118864	N	31.6	12.4	6	147	5.12	132	4.2	97	-	-	-	1.09	201	40
37	492838	N	12.1	7.2	252	50	2.32	131	3.93	99	7.1	1.55	3.91	0.31	21	58
38	1122943	N	8.5	8.1	32	147	4.15	132	5.74	106	8	2	5.2	0.64	18	20
39	45946	N	20	11.4	293	93	2.25	135	3.98	90	8.1	1.6	7.4	1.03	30	33
40	1173418	N	16	10.9	166	70	0.69	149	3.96	115	7	2.14	2.64	1.33	50	45
41	1171335	N	12	9	120	62	1.13	141	3.32	97	6.4	1.37	6.96	1.82	66	26
42	1113188	N	16	6.1	268	256	8.13	112	6.12	82	9.6	2.37	3.95	0.35	16	16
43	891124	N	10.8	11.3	245	55	1.44	137	5.06	99	8.7	1.76	4.44	0.56	34	34
44	1169277	N	9.1	14.1	150	14	0.85	144	4.56	102	8.4	2.31	2.77	0.42	21	12
45	1167714	Y	33	12.8	39	61	3.33	132	5.67	97	7.3	-	-	7.31	1777	1003
46	1048315	N	9.5	11.2	223	101	3.22	138	3.48	100	8.4	2.1	3.02	1.11	19	8
47	1164650	N	18	13.6	465	45	0.95	138	4.7	95	8.9	4.29	8.68	0.18	58	31
48	1163484	N	23	10.9	248	51	1.27	148	4.31	120	10.1	2.26	3.2	0.38	55	58
49	1163390	N	21.1	15.2	177	97	0.99	150	4.18	109	10.2	2.67	4.33	3.35	87	132

S. No	MRD	ALP (U/L)	TOTAL PROTIENS (gm/dl)	S ALBUMIN (gm/dl)	ESR	CRP (mg/L)	PCT	HBV/ HCV/ HIV STATUS	B-1,3 GLUCAN ASSAY	GALACTOMANAN	ANTI-SARS COV2 ANTIBODIES	DENGUE/LEPTO/SCRUB	PROTEINS	GLUCOSE	RBC'S	PUS CELL
1	1308834	76	6.7	3.58	48	-	12.11	NR	68.5			-	1+	NEG	10-15	5
2	1314039	160	4.2	1.32	2	-	1.7	-	-	-	-	-	NEG	3+	NEG	NEG
3	999518	92	8.1	4.11	81	-	30.79	NR	-	-	-	-	3+	NEG	10-12	NEG
4	1315468	66	6.9	4.62	19	-	0.11	-	-	-	-	-	3+	NEG	NEG	NEG
5	1198413	664	5.7	2.34	38	-	-	NR	-	-	-	-	2+	NEG	3-4	65
6	945977	280	4.7	1.99	99	-	29	NR	49.52	0.19	-	-	1+	NEG	NEG	NEG
7	1312440	140	5.7	2.55	57	-	1.03	NR	97	0.46	-	-	3+	2+	8-10	NEG
8	1308883	108	7.6	3.42	30	-	0.71	NR	-	1.25	-	-	NEG	NEG	NEG	NEG
9	1308751	475	5.8	2.39	94	-	25.5	NR	-	-	-	-	2+	NEG	NEG	NEG
10	688165	140	6.5	2.86	57	66	1.12	NR	-	-	-	-	3+	NEG	NEG	NEG
11	1316788	143	7.9	5.55	2	-	-	NR	-	-	-	-	NEG	1+	8-10	NEG
12	1318291	85	6	3.42	44	-	3.54	NR	-	-	-	-	-	-	-	-
13	1312474	80	6.3	3.75	10	-	10.38	NR	-	-	-	-	1+	NEG	0-2	NEG
14	1317402	83	6.4	3.03	22	24	1.84	NR	-	-	-	-	1+	NEG	NEG	NEG
15	1313135	82	6.9	3.79	65	-	-	NR	-	-	-	-	3+	NEG	NEG	NEG
16	1317416	91	4.6	2.4	18	92	32.49	NR	-	-	-	-	TRACES	NEG	1-2	2
17	1313289	109	5.1	2.53	35	-	36.85	HCV REACTIVE	-	-	-	-	1+	2+	28-30	2
18	1313327	53	6.7	3.43	20	37.91	0.07	NR	-	-	-	-	TRACES	NEG	NEG	NEG
19	1311126	164	5.8	2.03	87	41	-	NR	25	0.1	1130	-	NEG	NEG	NEG	NEG
20	1229583	168	5.5	1.82	74	-	0.07	NR	-	-	-	-	3+	NEG	40-50	NEG
21	1313107	143	5.1	2.22	113	147	0.98	NR	523	0.06	-	-	2+	NEG	26-28	10
22	1312496	97	6.3	3.81	21	-	6.26	-	-	-	-	-	2+	NEG	30-32	25
23	1119943	92	6.7	2.15	81	184	2.84	NR	73.23	0.73	23.52	-	-	-	-	-
24	1134066	68	6.3	3.29	7	-	10.88	-	-	-	-	-	3+	NEG	FULL FIELD	NEG
25	1236486	73	5.8	3.5	20	11.52	0.13	NR	20	0.1	-	-	NEG	NEG	NEG	NEG
26	1234438	79	6.9	3.75	47	29	16	NR	107	0.31	-	-	1+	NEG	NEG	NEG
27	1204887	251	6.6	3.4	3	-	0.53	-	-	-	-	-	NEG	1+	18-20	4
28	1266238	62	5.5	3.31	12	-	1.4	-	-	-	-	-	2+	NEG	30-40	NEG
29	1265486	82	5.3	2.54	-	231	18	-	-	-	-	-	2+	NEG	15-20	15
30	1131733	93	4.3	1.92	24	-	10	-	-	-	-	-	3+	TRACES	5-10	35
31	966910	79	5.6	2.82	82	334	16.68	NR	-	-	-	-	TRACES	NEG	0	15
32	445706	78	5.6	3.22	49	244	-	NR	125	0.1	-	-	2+	NEG	25-30	8
33	1210090	104	6.8	3.18	50	147	0.22	NR	63	3.87	5.23	DENGUE IGM +	2+	NEG	4-6	NEG
34	1118983	62	6.1	3.1	56	131	0.21	NR	172	0.1	0.19	LEPTO IGM +	2+	NEG	4.5	NEG
35	1118810	106	5.5	2.67	-	241	17.96	NR	-	-	-	-	2+	NEG	20-25	30
36	1118864	72	4.4	2.12	-	196	13.9	NR	-	-	-	-	3+	NEG	FULL FIELD	NEG
37	492838	165	4.9	1.95	50	116	0.23	NR	-	1.31	8.56	-	2+	NEG	OCCASSIONAL	NEG
38	1122943	249	6.1	2.34	70	260	1.85	NR	-	-	16.77	-	2+	NEG	6-7	100
39	45946	75	7.3	4.11	29	-	2.12	-	-	-	-	-	3+	NEG	10-20	NEG
40	1173418	38	4.7	2.84	13	4.59	0.17	NR	-	0.49	-	-	1+	NEG	NEG	5
41	1171335	290	5.8	2.33	40	-	0.32	NR	-	-	-	-	TRACES	NEG	1-2	4
42	1113188	226	6.2	2.95	120	-	3.38	NR	157	0.54	-	-	1+	NEG	10-12	90
43	891124	155	6.3	3.85	35	-	12.25	NR	-	-	-	-	TRACES	3+	NEG	NEG
44	1169277	62	6.2	4.01	3	0.68	0.12	NR	-	-	-	-	TRACES	3+	NEG	NEG
45	1167714	158	4.3	2.31	2	-	100	NR	-	-	-	-	2+	NEG	55-60	NEG
46	1048315	164	7	4.09	68	29	0.16	NR	-	-	-	-	3+	NEG	20-30	10
47	1164650	121	7.7	4.12	51	21	0.09	NR	-	-	-	-	1+	NEG	3-4	NEG
48	1163484	249	6.5	3.45	53	55.67	0.21	-	-	-	-	-	1+	NEG	8-10	18
49	1163390	291	5.8	4.04	3	-	0.14	NR	-	0.57	-	-	2+	NEG	NEG	NEG

S. No	MRD	CULTURE GROWTH_URINE	BLOOD	CSF	SPUTUM/ET SECREATION	PUS/WOUND SWAB	PLEURAL FLUID	ASCITIC FLUID	2D ECHO LVEF	EF	GHK/RWMA	LVID (S)	LVID (D)
1	1308834	NEG	NEG	NEG	-	NEG	-	-	45	3	GHK	40	54
2	1314039	NEG	NEG	-	-	-	-	-	22	2	RWMA	48	54
3	999518	NEG	NEG	-	-	-	-	-	25	2	GHK	47	57
4	1315468	NEG	NEG	-	ACINETOBACTER,KLEBSIELLA	-	-	-	34	2	GHK	43	44
5	1198413	PSEUDOMONAS,ENTEROCOCCUS	-	-	-	-	-	-	20	2	RWMA	48	58
6	945977	CANDIDA	KLEBSIELLA	-	KLEBSIELLA	-	-	-	42	3	GHK	30	47
7	1312440	PROVIDENCIA	NEG	NEG	-	NEG	-	-	24	2	RWMA	51	59
8	1308883	-	-	-	-	NEG	-	-	45	3	RWMA	47	58
9	1308751	NEG	NEG	-	KLEBSIELLA	-	-	-	40	2	RWMA	53	73
10	688165	NEG	-	-	-	-	-	-	25	2	GHK	39	52
11	1316788	NEG	-	-	NEG	-	-	-	35	2	GHK	19	30
12	1318291	NEG	NEG	-	NEG	-	-	-	44	3	GHK	32	43
13	1312474	NEG	STAPH HEMOLYTICUS	-	NEG	NEG	-	-	44	3	GHK	39	50
14	1317402	CANDIDA	NEG	-	KLEBSIELLA	-	-	-	22	2	RWMA	52	62
15	1313135	CANDIDA	-	-	NEG	-	-	-	28	2	RWMA	45	55
16	1317416	NEG	NEG	-	NEG	-	-	-	22	2	GHK	38	55
17	1313289	E.COLI	KLEBSIELLA	-	-	-	-	-	28	2	RWMA	53	61
18	1313327	-	-	-	NEG	-	-	-	36	2	GHK	30	47
19	1311126	NEG	NEG	-	-	-	-	-	22	2	GHK	51	57
20	1229583	E.COLI	NEG	-	-	-	-	-	28	2	RWMA	50	60
21	1313107	CANDIDA	ENTEROCOCCUS	-	-	-	-	-	32	2	RWMA	44	56
22	1312496	ENTEROCOCCUS	NEG	-	PSEUDOMONAS, KLEBSIELLA	-	-	-	28	2	GHK	30	35
23	1119943	NEG	E.COLI	-	E.COLI	-	NEG	-	44	3	RWMA	38	49
24	1134066	NEG	SERRATIA MARCES.	-	ACINETOBACTER, KLEBSIELLA	-	-	-	40	2	RWMA	34	48
25	1236486	NEG	NEG	-	NEG	-	-	-	36	2	GHK	48	58
26	1234438	NEG	NEG	-	ACINETOBACTER, KLEBSIELLA	-	-	-	35	2	GHK	30	50
27	1204887	NEG	NEG	-	-	-	-	-	26	2	RWMA	10	46
28	1266238	NEG	NEG	-	NEG	-	-	-	16	1	RWMA	26	46
29	1265486	NEG	NEG	-	-	-	-	-	36	2	GHK	47	57
30	1131733	E.COLI	-	-	KLEBSIELLA	-	-	-	27	2	GHK	47	57
31	966910	NEG	NEG	-	-	-	-	-	34	2	GHK	47	62
32	445706	NEG	NEG	-	-	-	-	-	38	2	GHK	42	52
33	1210090	NEG	NEG	-	-	-	-	-	35	2	GHK	42	55
34	1118983	KLEBSIELLA	NEG	-	-	-	-	-	25	2	GHK	47	54
35	1118810	NEG	CANDIDA	-	-	-	-	-	40	2	GHK	44	60
36	1118864	NEG	NEG	-	-	-	-	-	48	3	GHK	27	46
37	492838	NEG	NEG	-	-	-	-	-	48	3	RWMA	32	43
38	1122943	KLEBSIELLA	KLEBSIELLA	-	-	KLEBSIELLA, PSEUDOMONAS	-	-	28	2	GHK	49	57
39	45946	NEG	-	-	-	-	-	-	16	1	GHK	48	58
40	1173418	-	NEG	-	-	-	-	-	32	2	RWMA	30	52
41	1171335	NEG	NEG	-	-	KLEBSIELLA	-	-	20	2	RWMA	45	50
42	1113188	E.COLI	NEG	-	-	-	-	-	42	3	GHK	37	47
43	891124	-	STAPH EPIDERMIDIS	-	-	-	-	-	38	2	GHK	51	59
44	1169277	-	-	-	-	-	-	-	44	3	GHK	34	46
45	1167714	CANDIDA	STAPH EPIDERMIDIS	-	-	-	-	-	16	1	GHK	41	47
46	1048315	NEG	NEG	-	-	-	-	-	33	2	RWMA	49	59
47	1164650	NEG	-	-	NEG	-	-	-	30	2	GHK	48	58
48	1163484	NEG	-	NEG	-	-	-	-	38	2	RWMA	42	52
49	1163390	NEG	NEG	-	ACINETOBACTER	-	-	-	45	3	GHK	33	37

S. No	MRD	PA PRESSURE (PASP)	PAO2/FIAO2	PLATELETS	TOTAL BILIRUBIN	MAP	GCS	S. CREATININE/ URINE OUTPUT	SOFA SCORE ON INCLUSION DATE	TOTAL HOSPITAL STAY DURATION (DAYS)	OUTCOME	STATUS OF PATIENT AT 30 DAYS AFTER DISCHARGE ALIVE	DEAD
1	1308834	40	3	0	2	4	3	2	14	23	DISCHARGE	Y	N
2	1314039	20	2	0	0	4	3	1	10	14	DISCHARGE	N	Y
3	999518	45	2	0	0	0	3	4	9	5	DAMA	N	Y
4	1315468	20	2	0	0	0	0	1	3	23	DEATH	N	Y
5	1198413	20	2	0	1	4	1	2	10	4	DEATH	N	Y
6	945977	20	1	1	0	4	3	2	11	19	DISCHARGE	Y	N
7	1312440	52	4	0	0	0	1	3	8	46	DEATH	N	Y
8	1308883	20	3	0	2	3	0	0	8	18	DISCHARGE	Y	N
9	1308751	40	2	0	2	3	0	2	9	26	DISCHARGE	N	Y
10	688165	50	3	0	0	0	2	4	9	11	DISCHARGE	N	Y
11	1316788	20	3	0	0	0	4	0	7	7	DISCHARGE	Y	N
12	1318291	50	3	0	0	0	3	4	10	6	DAMA	N	Y
13	1312474	20	0	0	2	0	0	4	6	28	DISCHARGE	Y	N
14	1317402	20	3	0	0	3	0	2	8	16	DISCHARGE	Y	N
15	1313135	20	4	0	0	0	0	4	8	24	DAMA	Y	N
16	1317416	30	4	0	0	3	1	0	8	9	DISCHARGE	Y	N
17	1313289	40	0	2	0	0	2	2	6	10	DAMA	N	Y
18	1313327	20	3	0	1	0	4	2	10	6	DEATH	N	Y
19	1311126	20	4	0	0	0	0	0	4	22	DEATH	N	Y
20	1229583	55	1	0	0	0	2	2	5	5	DAMA	Y	N
21	1313107	20	3	2	0	0	1	4	10	7	DAMA	N	Y
22	1312496	35	0	1	0	0	1	1	3	13	DAMA	N	Y
23	1119943	20	2	0	0	0	0	0	2	14	DAMA	N	Y
24	1134066	30	0	3	1	3	3	2	12	9	DEATH	N	Y
25	1236486	20	2	0	1	0	3	0	6	12	DISCHARGE	Y	N
26	1234438	20	3	1	0	3	2	4	13	16	DAMA	N	Y
27	1204887	20	0	0	1	3	0	1	5	6	DISCHARGE	N	Y
28	1266238	20	4	0	0	4	3	2	13	5	DAMA	N	Y
29	1265486	40	1	2	3	3	0	3	12	15	DAMA	N	Y
30	1131733	20	2	0	0	4	4	4	14	16	DAMA	N	Y
31	966910	30	0	1	0	3	3	2	9	3	DEATH	N	Y
32	445706	20	2	0	2	3	0	2	9	10	DISCHARGE	Y	N
33	1210090	20	4	0	0	3	0	0	7	30	DISCHARGE	Y	N
34	1118983	20	4	2	2	1	0	4	13	18	DISCHARGE	Y	N
35	1118810	40	4	1	2	4	2	4	17	18	DAMA	Y	N
36	1118864	20	3	3	0	2	1	4	13	10	DISCHARGE	Y	N
37	492838	30	3	0	0	2	1	3	9	23	DISCHARGE	Y	N
38	1122943	64	0	3	0	1	1	3	8	8	DAMA	N	Y
39	45946	50	0	0	0	3	1	1	5	9	DISCHARGE	Y	N
40	1173418	50	2	0	1	3	2	0	8	11	DISCHARGE	Y	N
41	1171335	40	2	2	1	3	2	0	10	24	DISCHARGE	Y	N
42	1113188	35	3	0	0	4	2	4	13	18	DAMA	N	Y
43	891124	60	3	0	0	2	0	1	6	6	DISCHARGE	Y	N
44	1169277	20	2	1	0	3	2	0	8	18	DISCHARGE	Y	N
45	1167714	30	3	3	3	4	2	2	17	4	DAMA	N	Y
46	1048315	35	4	2	2	4	4	4	20	7	DEATH	N	Y
47	1164650	20	1	0	0	3	3	1	8	12	DISCHARGE	Y	N
48	1163484	20	3	0	0	3	1	1	8	9	DAMA	Y	N
49	1163390	20	2	0	2	4	4	0	12	7	DAMA	N	Y

S. No	MRD	INITIALS	AGE	AGE GROUP	SEX	DOA	DOD	FEVER	COUGH/ SPUTUM	PAIN ABDOMEN	DECREASED URINE OUTPUT	BURNING/ INCEASED FREQUENCY OF URINATION	LOOSE STOOLS	ALTRED SENSORIUM/ SIEZURES	SOB	VOMITING/ NAUSEA	OTHERS	DM	HTN	COPD/B.ASTHMA/ANY RESP DISORDER	CLD
50	1164122	RS	35	2	M	30-08-2021	02-09-2021	Y	N	N	N	N	N	Y	N	N	HEADACHE	Y	N	N	N
51	1064866	RJ	65	5	M	31-08-2021	17-09-2021	Y	N	Y	Y	N	N	Y	N	N	N	Y	Y	N	N
52	1162712	JS	42	3	M	28-08-2021	04-09-2021	N	Y	N	N	N	N	N	Y	N	LETHARGY	N	Y	N	N
53	1128898	SK	72	6	F	26-08-2021	04-09-2021	Y	N	N	N	N	N	Y	N	N	CONSTIPATION	N	Y	N	N
54	836700	BK	53	4	F	02-08-2021	01-09-2021	Y	N	Y	N	Y	N	N	N	N	N	Y	Y	N	N
55	1158354	S	18	1	M	21-08-2021	11-09-2021	Y	Y	N	N	N	N	N	N	N	N	Y	N	N	N
56	1156398	AM	60	4	M	18-08-2021	23-08-2021	N	Y	N	N	N	N	Y	Y	N	N	N	Y	N	N
57	884071	HL	81	6	M	14-08-2021	20-08-2021	Y	N	N	Y	N	N	Y	Y	N	N	Y	Y	N	N
58	1157068	RK	52	4	F	19-08-2021	10-09-2021	Y	N	N	N	N	N	N	Y	N	N	Y	N	N	N
59	1142597	RB	65	5	F	25-07-2021	11-08-2021	Y	N	N	N	N	N	N	Y	N	CHEST PAIN	N	Y	Y	N
60	1150441	P	25	1	M	07-08-2021	25-08-2021	N	N	N	Y	N	N	Y	N	Y	N	N	N	N	N
61	1150193	JS	33	2	M	07-08-2021	20-08-2021	Y	N	N	N	N	N	N	Y	N	N	N	N	N	N
62	1125632	AS	79	6	M	31-07-2021	13-08-2021	Y	N	Y	N	N	N	N	N	N	N	N	Y	N	N
63	1132230	JS	60	4	M	06-08-2021	21-08-2021	N	N	N	N	N	N	N	N	N	PERI-ORBITAL SWELLING	Y	N	N	N
64	1146512	AK	65	5	M	27-10-2021	28-10-2021	Y	N	N	Y	Y	N	Y	N	N	N	Y	Y	N	N
65	1142584	NR	40	2	F	25-07-2021	31-07-2021	N	N	N	Y	N	N	N	Y	Y	PEDAL SWELLING	N	N	N	N
66	1108226	MK	60	4	F	13-05-2021	30-06-2021	N	N	N	N	N	N	N	N	N	PERI-ORBITAL SWELLING	Y	N	N	N
67	1089518	KD	58	4	F	19-04-2021	15-05-2021	Y	Y	N	N	N	N	N	Y	N	N	Y	N	N	N
68	1100518	SK	55	4	F	01-05-2021	13-05-2021	N	N	N	Y	N	N	Y	N	N	ABDOMEN DISTENSION	N	Y	N	N
69	811219	RK	66	5	M	28-04-2021	13-05-2021	N	N	N	Y	N	Y	Y	N	N	N	Y	Y	N	N
70	1097530	AB	58	4	F	02-05-2021	06-05-2021	Y	N	N	N	N	Y	Y	N	N	N	Y	Y	Y	N
71	1078473	RK	88	6	F	23-04-2021	05-05-2021	Y	N	Y	N	N	N	N	N	N	N	Y	N	N	N
72	1088794	BR	66	5	M	10-06-2021	15-06-2021	N	Y	N	N	Y	N	N	Y	N	N	Y	Y	N	N
73	1132967	MB	47	3	F	14-07-2021	03-08-2021	Y	N	Y	N	N	N	N	N	Y	N	N	Y	N	N
74	709606	JS	66	5	M	08-02-2021	08-07-2021	Y	N	N	Y	Y	N	N	N	N	N	Y	Y	N	Y
75	102328	RK	67	5	F	24-06-2021	26-07-2021	N	Y	Y	Y	Y	N	Y	Y	N	PEDAL SWELLING	Y	Y	N	N
76	1016955	GR	79	6	F	24-01-2022	28-01-2022	Y	N	N	Y	Y	N	N	Y	N	N	Y	Y	N	N
77	1088286	HS	51	4	M	17-04-2021	26-04-2021	Y	Y	N	N	N	N	N	Y	N	N	Y	N	N	N
78	1090724	AK	21	1	F	20-04-2021	24-04-2021	Y	N	N	N	Y	N	N	Y	N	PER VAGINUM BLEED	N	N	N	N
79	232443	JS	79	6	M	10-04-2021	24-04-2021	N	N	N	N	N	N	Y	N	N	N	Y	Y	N	N
80	1071146	SR	48	3	F	01-04-2021	09-04-2021	N	Y	N	N	N	N	N	Y	N	N	Y	Y	N	N
81	560295	RR	64	5	F	31-01-2022	05-02-2022	N	Y	N	N	N	N	N	Y	Y	N	Y	Y	N	N
82	1022005	JK	35	2	F	03-02-2022	12-02-2022	Y	N	Y	N	N	N	Y	N	Y	N	N	N	N	N
83	1005243	SKS	71	6	M	03-02-2022	23-02-2022	Y	Y	N	N	N	N	N	Y	Y	N	N	Y	N	N
84	975534	TS	61	5	M	28-01-2022	17-02-2022	Y	N	N	N	Y	N	N	N	N	N	Y	Y	N	N
85	1020128	PA	55	4	F	31-01-2022	06-02-2022	N	Y	N	N	Y	N	N	Y	N	CHEST PAIN	Y	Y	N	N
86	1018513	HK	52	4	M	28-01-2022	10-02-2022	Y	Y	N	N	N	N	N	N	N	N	Y	N	N	N
87	1019045	HS	25	1	M	28-01-2022	04-02-2022	N	N	N	N	N	N	Y	N	N	N	N	N	N	N
88	984239	RSP	53	4	M	23-01-2022	02-02-2022	N	Y	N	N	N	N	N	Y	N	N	Y	N	N	N
89	1023853	S	48	3	M	06-02-2022	03-03-2022	N	N	N	N	N	N	Y	N	Y	BLOOD IN URINE	N	N	N	Y
90	576311	KS	67	5	M	07-04-2021	18-04-2021	N	N	N	N	Y	N	N	N	N	SLURRED SPEECH, DIZZINESS	N	Y	N	N
91	1081594	TR	57	4	M	28-04-2021	05-05-2021	N	Y	N	N	N	N	N	N	N	CHEST PAIN	Y	N	N	N
92	1084339	PK	56	4	M	13-04-2021	01-05-2021	N	N	N	N	N	N	N	N	N	FOOT GANGRENE	Y	Y	N	N
93	1087151	MK	87	6	F	16-04-2021	28-04-2021	N	N	Y	Y	N	N	Y	N	N	N	Y	Y	N	N
94	1067817	TC	75	6	M	29-03-2021	11-04-2021	N	N	N	Y	N	N	Y	Y	N	N	Y	Y	N	N
95	669637	CL	69	5	M	10-07-2021	02-08-2021	Y	Y	N	N	N	N	N	Y	N	N	Y	Y	N	N
96	1024042	SC	62	5	M	07-02-2022	24-02-2022	N	Y	N	N	N	N	Y	Y	N	N	Y	Y	Y	N

S. No	MRD	CKD	CAD	HYPOTHYROIDISM	CVA	OTHER MEDICAL HISTORY	SURGICAL HISTORY	ALCHOL	SMOKING	OTHER SUBSTANCE	GCS	gcs GROUP	SBP	DBP	HR	RR	SPO2%	FIO2 %	RTI	UTI	GIT	SKIN	BLOOD STREAM	CNS-INFECTION
50	1164122	N	N	N	N	N	N	Y	Y	OPIOID, TOBACCO	8	1	100	60	76	16	94	90	Y	Y	N	N	N	N
51	1064866	N	N	N	N	N	N	N	N	N	5	1	70	40	90	16	88	100	N	Y	N	N	Y	Y
52	1162712	Y	N	N	N	N	N	Y	N	N	15	3	140	90	92	30	89	50	Y	N	N	N	N	N
53	1128898	N	N	Y	Y	PARKINSON	N	N	N	N	6	1	60	30	86	24	85	21	N	Y	Y	N	N	N
54	836700	Y	N	N	N	N	DJ STENTING	N	N	N	15	3	110	70	117	18	94	21	N	Y	N	N	N	N
55	1158354	N	N	N	N	N	N	N	N	N	15	3	110	70	116	20	92	21	Y	N	N	N	N	N
56	1156398	N	N	N	N	N	N	Y	Y	N	10	2	120	70	89	22	92	100	N	N	N	N	N	N
57	884071	Y	Y	N	N	DEMENTIA	N	N	N	N	12	2	90	60	104	24	85	21	N	Y	N	N	N	N
58	1157068	N	N	-	N	N	N	N	N	N	3	1	100	70	117	24	68	50	Y	N	N	N	Y	N
59	1142597	N	N	N	N	N	CHOLECYSTECTOMY, HERNIA SURGERY	N	N	N	15	3	90	60	131	36	92	100	Y	N	N	N	N	N
60	1150441	N	N	N	N	N	N	N	N	N	5	1	90	60	117	16	96	50	Y	N	N	N	N	N
61	1150193	N	N	N	N	POST COVID	N	N	N	N	14	3	130	70	124	26	94	90	Y	N	N	N	N	N
62	1125632	N	N	N	N	POST COVID	POST LAPAROTOMY	N	N	N	15	3	100	70	84	18	93	21	Y	Y	Y	N	N	N
63	1132230	N	N	N	N	POST COVID	N	N	N	N	15	3	110	70	164	16	96	21	Y	N	N	N	N	N
64	1146512	N	N	N	N	DEPRESSIVE ILLNESS	N	N	Y	N	8	1	60	30	111	25	74	21	N	Y	N	N	Y	N
65	1142584	N	N	N	N	N	HYSTERECTOMY	N	N	N	15	3	100	70	107	30	94	100	N	Y	N	N	N	N
66	1108226	N	N	N	N	POST COVID	N	N	N	N	15	3	120	70	97	18	92	21	Y	N	N	N	N	N
67	1089518	N	N	Y	N	N	N	N	N	N	15	3	110	70	123	32	94	50	Y	N	N	N	N	N
68	1100518	N	N	Y	N	N	N	N	N	N	11	2	130	70	130	28	96	100	N	Y	N	N	N	N
69	811219	Y	N	N	N	N	BIG TOE AMPUTATION	N	Y	N	10	2	60	30	134	30	60	21	Y	Y	Y	Y	N	N
70	1097530	N	N	N	Y	N	N	N	N	N	8	1	60	30	117	28	65	21	N	N	Y	N	N	N
71	1078473	N	N	N	N	N	CHOLECYSTECTOMY	N	N	N	15	3	130	70	100	18	96	33	N	Y	N	N	Y	N
72	1088794	N	Y	Y	N	POST COVID	N	Y	Y	N	15	3	130	70	96	20	92	50	Y	Y	N	N	N	N
73	1132967	N	N	Y	N	N	N	N	N	N	15	3	110	80	112	26	92	21	N	Y	N	N	N	N
74	709606	N	Y	N	N	N	POST CABG	Y	N	N	14	3	100	80	80	20	96	29	Y	Y	N	N	N	N
75	102328	Y	N	Y	N	N	N	N	N	N	10	2	80	60	100	28	96	100	N	Y	N	N	N	N
76	1016955	N	Y	Y	N	N	CHOLECYSTECTOMY	N	N	N	14	3	100	60	112	18	94	50	Y	Y	N	N	N	N
77	1088286	N	N	N	N	N	N	Y	N	N	15	3	110	70	117	26	86	100	Y	N	N	Y	N	N
78	1090724	N	N	N	N	N	N	N	N	N	11	2	80	40	127	28	79	21	N	Y	N	N	N	N
79	232443	N	Y	N	N	PARKINSON	POST NEPHRECTOMY	Y	Y	N	9	2	90	60	111	18	88	21	N	Y	N	N	N	N
80	1071146	N	N	N	N	N	N	N	N	N	15	3	90	60	117	28	92	100	Y	N	N	N	N	N
81	560295	N	Y	N	N	N	N	N	N	N	15	3	110	70	106	28	93	50	Y	N	N	N	N	N
82	1022005	N	N	N	N	N	N	N	N	N	12	2	60	30	112	22	94	21	N	N	N	N	N	N
83	1005243	N	N	N	N	MOTOR NEURON DISEASE	POST TRACHEOSTOMY	N	N	N	9	2	100	60	104	24	80	60	Y	N	N	Y	Y	N
84	975534	N	Y	N	N	DEPRESSIVE ILLNESS	POST DJ STENTING	N	N	N	15	3	100	60	96	18	92	21	N	Y	N	N	N	N
85	1020128	N	N	N	N	N	N	N	N	N	15	3	130	70	102	26	86	21	N	Y	N	N	N	N
86	1018513	N	N	N	N	N	N	Y	Y	N	15	3	110	70	99	26	94	100	Y	N	N	N	N	N
87	1019045	N	N	N	N	N	N	Y	Y	OPIOID, COCAINE	4	1	60	30	127	14	69	21	Y	N	N	N	N	N
88	984239	N	N	N	N	POST RSA-HEAD INJURY	POST CRANIECTOMY	N	N	N	10	2	90	60	131	26	86	50	Y	Y	N	N	N	N
89	1023853	N	N	N	N	N	N	Y	N	N	6	1	110	70	94	18	100	100	Y	N	N	N	Y	N
90	576311	N	Y	N	N	N	POST CABG	Y	N	N	12	2	100	70	90	21	88	21	N	Y	N	N	N	N
91	1081594	N	Y	N	N	N	N	N	Y	N	15	3	90	60	124	30	94	50	Y	N	N	N	N	N
92	1084339	N	Y	N	N	N	N	N	N	N	15	3	110	70	99	20	96	21	Y	N	N	Y	Y	N
93	1087151	N	N	N	N	N	N	N	N	N	11	2	90	70	113	28	92	50	N	Y	N	N	Y	N
94	1067817	N	N	N	N	BURNS, HERNIA, BED RIDDEN	N	Y	Y	N	11	2	80	60	117	28	93	50	Y	Y	N	Y	N	N
95	669637	N	Y	N	N	CA PROSTATE	POST CABG, POST TURP	N	Y	N	15	3	110	70	112	28	93	80	Y	N	N	N	N	N
96	1024042	Y	N	N	N	N	N	N	Y	N	8	1	80	60	124	28	72	21	Y	N	N	N	N	N

S. No	MRD	GENITAL INFECTION	TLC (X 1000/ UL)	HB (gm/dl)	PLATELETS (X 1000/ UL)	UREA (mg/dl)	CREATININE (mg/dl)	SERUM SODIUM (mmol/L)	S. POTASSIUM (mmol/L)	S. CHLORIDE (mmol/L)	S. CALCIUM	S. MAGNESIUM	S. PHOSPHORUS	TOTAL BILIRUBIN (mg/dl)	SGOT (U/L)	SGPT (U/L)
50	1164122	N	8.7	12.8	180	25	0.37	134	3.32	101	8.4	1.93	2.4	1.4	63	28
51	1064866	N	19.3	13.6	160	96	2.78	151	3.58	111	7.2	1.97	3.17	0.23	1533	1271
52	1162712	N	10.8	8.5	275	83	8.8	136	3.34	98	8	2.83	1.48	0.61	27	17
53	1128898	N	22	8.5	381	123	1.01	136	3.94	91	8.9	2.36	2.19	0.54	103	18
54	836700	N	19	7.7	316	110	7.06	130	4.81	99	9.3	2.01	2.6	0.46	35	10
55	1158354	N	10.4	11.4	200	39	0.79	144	3.7	113	7.7	1.74	2	0.59	38	26
56	1156398	N	13.3	14.1	191	38	1.23	141	5.01	107	8.5	2.54	5.77	0.64	25	29
57	884071	N	10.4	12.1	140	214	7.52	127	3.38	92	8.5	2.22	5.1	4.19	176	178
58	1157068	N	11.5	6.9	121	163	7.82	126	5.1	86	7.5	1.9	4.26	0.34	30	36
59	1142597	N	10.8	10.4	177	76	1.52	140	4.46	97	7.7	1.45	3.8	0.3	78	38
60	1150441	N	10.4	7.8	257	89	7.08	136	4.98	97	9.6	3.28	4.4	0.33	25	35
61	1150193	N	11.4	11.7	234	29	0.74	143	3.87	104	7.8	2.18	2.61	0.51	45	27
62	1125632	N	5.4	9.2	349	30	1.07	134	3.39	97	7	1.4	3.3	0.32	22	16
63	1132230	N	11.8	10.8	373	41	1.69	139	4.78	102	9.7	2.04	2.16	0.2	13	11
64	1146512	N	20	7.8	192	114	2.32	104	3.14	69	6.2	1.42	2.59	1.1	55	49
65	1142584	N	21	8.4	561	108	5.95	136	3.95	99	8.4	2.16	7	0.7	17	3
66	1108226	N	6.8	8	458	19	1.26	133	3.6	109	7.1	1.33	2.2	0.7	27	28
67	1089518	N	11.3	10.8	216	50	0.8	140	3.7	107	7.6	2.25	2.82	0.06	44	33
68	1100518	N	11.5	8.9	80	42	2.29	142	4.69	114	7.5	2.09	4.35	1.1	41	17
69	811219	N	11.9	11.5	322	288	5.96	127	4.5	100	8.3	1.98	5.64	0.19	14	2
70	1097530	N	8.6	9.1	26	130	1.77	8.2	4.06	90	8.2	1.8	2.25	1.26	51	43
71	1078473	N	9.1	10.2	88	176	3.04	130	4.3	96	7.8	1.4	1.3	1.03	66	36
72	1088794	N	7.7	11.8	212	14	0.6	139	3.9	100	-	-	-	0.28	158	232
73	1132967	N	15.1	8.5	151	77	2.9	137	3.1	101	8.1	1.57	5.5	0.2	209	52
74	709606	N	17	10	154	109	2.3	113	3.4	87	8.5	2.8	6.8	18.3	189	355
75	102328	N	6	9.3	143	240	3.1	135	3.5	96	8.3	2.2	6.2	1.5	22	13
76	1016955	N	7.4	5.2	197	121	1.94	137	7.23	109	7.1	2.22	5.8	0.1	33	11
77	1088286	N	25	7.5	283	20	1.11	145	4.43	111	8.7	1.77	0.56	0.6	21	13
78	1090724	Y	13	5.9	783	75	2.33	139	4.29	104	8.3	2.39	5	0.56	25	11
79	232443	N	9.3	10.1	232	159	4.06	126	5.76	90	7.4	2.36	9.3	0.48	21	3
80	1071146	N	12.8	11.5	255	88	1.9	134	6.88	102	8.9	1.81	6.06	0.33	17	21
81	560295	N	10	9.1	351	224	3.36	136	6.65	98	9.9	1.93	5.63	0.4	70	83
82	1022005	Y	13.6	7.2	191	67	1.61	139	4.57	102	8.3	1.27	5.41	3.35	54	52
83	1005243	N	18	8.4	120	30	0.69	137	3.54	106	8.4	1.61	2.3	0.67	16	7
84	975534	N	4.9	8.5	144	123	5.06	141	3.57	96	9.1	1.36	3.9	0.07	24	21
85	1020128	N	19.8	10.5	345	42	0.74	129	6.8	98	8.2	1.97	3.3	0.04	13	10
86	1018513	N	12.2	12.6	437	47	0.98	130	6.16	93	8.6	2.13	3.03	0.18	40	27
87	1019045	N	16.8	14	416	33	0.74	141	4.28	108	8.4	2.62	11.11	0.33	284	221
88	984239	N	11	13.6	292	12	0.48	131	5	92	10.2	1.99	3.62	0.31	40	50
89	1023853	N	19.5	10.3	66	55	0.84	139	2.66	90	6.6	4.02	2.4	7.9	77	82
90	576311	N	7.8	12.2	203	68	1.5	143	4.37	107	9.2	1.9	3.76	0.79	17	14
91	1081594	N	3.2	10.7	70	40	1.13	121	3.91	86	8.2	1.9	4.13	0.87	115	303
92	1084339	N	16.3	9.3	158	161	1.73	148	5.51	120	9	2.65	2.74	0.9	18	18
93	1087151	N	23	8.3	418	108	3.46	127	5.53	93	7.6	1.07	4.6	0.48	23	15
94	1067817	N	21	7.4	110	141	2.13	135	7.35	99	8.2	2.17	5.31	0.36	1865	1130
95	669637	N	5.4	10.5	130	27	1.07	135	5.35	102	8	2.02	4.64	0.45	43	25
96	1024042	N	18.8	7.7	274	202	6.58	130	5.86	99	8.2	1.7	8.8	0.33	9	2

S. No	MRD	ALP (U/L)	TOTAL PROTIENS (gm/dl)	S ALBUMIN (gm/dl)	ESR	CRP (mg/L)	PCT	HBV/ HCV/ HIV STATUS	B-1,3 GLUCAN ASSAY	GALACTOMANAN	ANTI-SARS COV2 ANTIBODIES	DENGUE/LEPTO/SCRUB	PROTEINS	GLUCOSE	RBC'S	PUS CELL
50	1164122	147	7.4	3.81	36	-	2.08	NR	-	-	-	-	1+	2+	48-50	4
51	1064866	122	5.2	2.72	52	-	19.83	-	56.45	0.15	-	-	1+	NEG	NEG	NEG
52	1162712	222	6.5	3.79	22	-	1.64	NR	-	-	-	-	3+	NEG	1-2	NEG
53	1128898	199	7.2	3.36	120	-	1.46	-	-	-	-	-	1+	NEG	5-8	30
54	836700	138	6.6	2.59	-	127	1.11	NR	-	-	-	-	3+	2+	2-4	12
55	1158354	178	5.3	2.37	101	231	1.24	NR	7.8	0.37	-	-	1+	1+	10-12	NEG
56	1156398	145	7.5	4.77	6	-	-	-	-	-	-	-	3+	1+	2-3	NEG
57	884071	266	6.3	3.52	47	172	12.88	NR	-	-	-	-	1+	NEG	4-6	12
58	1157068	62	7.3	3.37	120	-	1.62	-	-	-	-	-	-	-	-	-
59	1142597	92	6.6	3.41	45	26	2.42	-	72	0.31	-	-	TRACES	NEG	6-8	4
60	1150441	49	6.3	3.54	15	-	2.19	NR	-	-	-	-	3+	NEG	15-18	2
61	1150193	167	5.8	2.92	26	99	2.59	HCV REACTIVE	126	0.12	-	-	2+	NEG	14-16	NEG
62	1125632	79	4.4	1.64	26	275	14.98	NR	523	0.08	-	-	1+	NEG	NEG	8
63	1132230	109	7.5	4.17	-	8.58	0.18	NR	-	-	-	-	TRACES	NEG	NEG	NEG
64	1146512	188	4	1.68	2	-	100	NR	-	-	-	-	1+	NEG	2-3	2
65	1142584	82	6.3	3.03	-	162	1.21	NR	-	-	-	-	TRACES	NEG	NEG	6
66	1108226	140	5.65	1.95	35	57.58	5	NR	-	-	62.88	-	TRACES	3+	NEG	NEG
67	1089518	142	6.2	2.94	-	35	0.69	NR	76.65	0.65	-	-	TRACES	NEG	NEG	8
68	1100518	233	5.1	3.23	11	-	0.14	NR	-	-	-	-	3+	NEG	20-22/HPF	100
69	811219	78	6.6	2.57	85	130.2	8	-	-	-	0.12	-	1+	NEG	12-16/HPF	8
70	1097530	147	5.5	3.42	2	66.66	0.9	-	137.25	-	-	-	NEG	NEG	NEG	NEG
71	1078473	120	6.3	3.11	103	294.83	63.31	-	-	-	-	LEPTO IGM+	3+	NEG	25-30/HPF	60
72	1088794	179	6.8	3.31	61	94.36	-	-	-	-	72.13	-	NEG	3+	NEG	25
73	1132967	357	7.4	2.36	-	41.54	1.04	NR	-	0.21	0.12	-	TRACES	NEG	2-3/HPF	5
74	709606	235	5	2.64	24	-	1.37	HBV REACTIVE	-	-	-	-	1+	NEG	>15-20/HPF	NEG
75	102328	153	7.1	3.41	34	10.25	0.29	NR	-	-	-	-	1+	NEG	2-3/HPF	3
76	1016955	102	6.6	3.3	50	3.96	19.42	NR	-	-	-	-	1+	TRACES	NEG	NEG
77	1088286	159	8	3.61	70	412	22.42	-	395	0.16	24.46	-	1+	3+	NEG	NEG
78	1090724	214	5.8	2.43	145	297	69.8	NR	215	0.44	11.71	-	3+	NEG	18-20/HPF	NEG
79	232443	139	6.4	3.51	88	-	41.86	NR	27.49	0.32	0.02	-	2+	NEG	40-45/HPF	50
80	1071146	145	7.8	3.93	78	10.15	1.88	-	-	-	0.03	-	1+	3+	4-6/HPF	NEG
81	560295	126	6.6	3.53	90	-	-	NR	-	-	-	-	1+	NEG	10-12/HPF	3
82	1022005	185	5.4	3.01	53	216	61.17	NR	96	0.3	21.8	LEPTO IGM+	1+	NEG	NEG	NEG
83	1005243	108	4.5	2.67	-	-	3.89	-	-	-	-	-	1+	NEG	NEG	4
84	975534	142	5.8	3.2	62	207	0.82	NR	-	0.03	-	-	3+	NEG	20-25/HPF	80
85	1020128	72	6.2	3.9	97	3.02	2.05	-	-	-	-	-	2+	3+	NEG	6
86	1018513	132	4.5	1.76	102	-	41.3	NR	165	0.42	-	-	1+	2+	20-25/HPF	NEG
87	1019045	108	5.9	3.47	14	36	1.7	HCV, HIV REACTIVE	-	-	-	-	1+	NEG	20-25/HPF	10
88	984239	106	7.6	4.64	26	-	0.75	-	-	-	-	-	3+	2+	NEG	10
89	1023853	92	6.6	2.9	-	109	9.38	-	279	-	0.137	-	NEG	NEG	2-4/HPF	NEG
90	576311	141	6.7	3.41	49	-	0.73	-	-	-	-	-	NEG	NEG	NEG	15
91	1081594	64	6.5	3.54	81	40	0.14	NR	-	-	-	-	NEG	NEG	NEG	NEG
92	1084339	165	5.3	2.56	70	156	3.52	NR	231	0.63	73.25	-	3+	NEG	30-40/HPF	NEG
93	1087151	61	5.4	2.45	60	-	100	-	187	0.9	2.18	-	3+	NEG	30-40/HPF	90
94	1067817	199	5.5	2.64	76	-	6.43	NR	-	0.51	0.03	-	-	-	-	-
95	669637	84	6	3.35	65	119	0.64	NR	80.01	0.39	44.32	-	TRACES	TRACES	4-6/HPF	NEG
96	1024042	98	6.9	2.59	120	163	3.99	NR	523	2.47	-	-	3+	1+	NEG	NEG

S. No	MRD	CULTURE GROWTH_URINE	BLOOD	CSF	SPUTUM/ET SECREATION	PUS/WOUND SWAB	PLEURAL FLUID	ASCITIC FLUID	2D ECHO LVEF	EF	GHK/RWMA	LVID (S)	LVID (D)
50	1164122	NEG	NEG	-	NEG	-	-	-	25	2	GHK	48	58
51	1064866	CANDIDA	CANDIDA	-	-	-	-	-	32	2	RWMA	33	44
52	1162712	NEG	NEG	-	-	-	-	-	24	2	GHK	37	48
53	1128898	CANDIDA	NEG	-	-	-	-	-	46	3	GHK	36	48
54	836700	ENTEROCOCCUS	-	-	-	-	-	-	48	3	GHK	39	48
55	1158354	NEG	NEG	-	ACINETOBACTER, KLEBSIELLA	-	-	-	28	2	GHK	42	58
56	1156398	NEG	-	-	ACINETOBACTER	-	-	-	25	2	GHK	44	60
57	884071	NEG	NEG	-	-	-	-	-	32	2	RWMA	46	56
58	1157068	-	STAPH EPIDERMIDIS	-	E.COLI	-	-	-	35	2	RWMA	46	54
59	1142597	-	-	-	-	-	-	-	32	2	GHK	41	50
60	1150441	NEG	NEG	NEG	-	-	-	-	35	2	GHK	34	41
61	1150193	NEG	-	-	-	-	-	-	45	3	GHK	42	60
62	1125632	PSEUDOMONAS, KLEBSIELLA	NEG	-	ACINETOBACTER	-	-	E.COLI	45	3	GHK	37	50
63	1132230	-	-	-	-	-	-	-	42	3	GHK	39	49
64	1146512	-	MORGANELLA	-	-	-	-	-	20	2	GHK	54	59
65	1142584	NEG	NEG	-	-	-	-	-	32	2	GHK	34	41
66	1108226	-	NEG	-	-	-	-	-	22	2	RWMA	26	46
67	1089518	-	-	-	ACINETOBACTER	-	-	-	34	2	RWMA	39	44
68	1100518	KLEBSIELLA	NEG	-	-	-	-	NEG	35	2	RWMA	39	52
69	811219	NEG	NEG	-	ACINETOBACTER, KLEBSIELLA	NEG	-	-	26	2	RWMA	44	51
70	1097530	NEG	NEG	-	-	-	-	-	32	2	GHK	43	49
71	1078473	E.COLI	E.COLI	-	-	-	-	-	25	2	GHK	40	54
72	1088794	-	-	-	NEG	-	-	-	40	2	RWMA	42	52
73	1132967	NEG	NEG	-	-	-	-	-	38	2	GHK	43	56
74	709606	NEG	-	-	KLEBSIELLA	-	-	NEG	48	3	RWMA	30	49
75	102328	KLEBSIELLA	-	-	-	-	-	-	20	2	GHK	46	51
76	1016955	-	NEG	-	NEG	-	-	-	16	1	GHK	26	46
77	1088286	NEG	NEG	-	NEG	NEG	-	-	36	2	GHK	37	49
78	1090724	ENTEROCOCCUS	NEG	-	NEG	-	-	-	45	3	GHK	30	46
79	232443	PSEUDOMONAS, PROVIDENCIA	-	-	-	-	-	-	40	2	RWMA	38	47
80	1071146	-	-	-	-	-	-	-	28	2	RWMA	32	50
81	560295	-	-	-	-	-	-	-	26	2	GHK	45	51
82	1022005	NEG	NEG	-	NEG	-	-	NEG	32	2	RWMA	39	50
83	1005243	NEG	KLEBSIELLA	-	ACINETOBACTER	ACINETOBACTER, KLEBSIELLA	-	-	38	2	RWMA	37	48
84	975534	KLEBSIELLA	-	-	-	-	-	-	36	2	GHK	41	52
85	1020128	NEG	-	-	-	-	-	-	32	2	RWMA	29	37
86	1018513	NEG	NEG	-	NEG	-	-	-	28	2	RWMA	44	51
87	1019045	-	NEG	NEG	ACINETOBACTER, KLEBSIELLA	-	-	-	35	2	RWMA	30	37
88	984239	NEG	-	-	NEG	-	-	-	36	2	GHK	30	50
89	1023853	-	KLEBIELLA	-	KLEBSIELLA	-	-	NEG	38	2	GHK	40	55
90	576311	NEG	NEG	-	-	-	-	-	18	1	RWMA	56	63
91	1081594	-	-	-	-	-	-	-	45	3	GHK	43	56
92	1084339	NEG	KLEBIELLA	-	-	KLEBSIELLA	-	-	24	2	GHK	35	40
93	1087151	-	E.COLI	-	-	-	-	-	48	3	GHK	35	44
94	1067817	CANDIDA	NEG	-	-	ACINETOBACTER, KLEBSIELLA, PSEUDOMONAS	-	-	22	2	RWMA	48	62
95	669637	NEG	NEG	-	NEG	-	-	-	36	2	RWMA	28	48
96	1024042	NEG	NEG	-	SERRATIA	-	-	-	40	2	GHK	52	63

S. No	MRD	PA PRESSURE (PASP)	PAO2/FIAO2	PLATELETS	TOTAL BILIRUBIN	MAP	GCS	S. CREATININE/ URINE OUTPUT	SOFA SCORE ON INCLUSION DATE	TOTAL HOSPITAL STAY DURATION (DAYS)	OUTCOME	STATUS OF PATIENT AT 30 DAYS AFTER DISCHARGE ALIVE	DEAD
50	1164122	20	3	0	1	3	3	0	10	4	DAMA	N	Y
51	1064866	40	3	0	0	4	4	2	13	18	DAMA	N	Y
52	1162712	60	4	0	0	3	0	4	11	8	DISCHARGE	Y	N
53	1128898	20	3	0	0	4	4	1	12	10	DAMA	N	Y
54	836700	20	2	4	0	3	1	4	14	30	DISCHARGE	Y	N
55	1158354	40	2	0	0	3	0	0	5	23	DISCHARGE	N	Y
56	1156398	20	3	0	0	3	2	1	9	6	DISCHARGE	Y	N
57	884071	40	2	1	2	4	4	4	17	7	DEATH	N	Y
58	1157068	40	3	1	0	3	4	4	15	23	DISCHARGE	N	Y
59	1142597	75	4	0	0	3	0	1	8	18	DISCHARGE	Y	N
60	1150441	20	2	0	0	0	4	4	10	19	DISCHARGE	Y	N
61	1150193	40	2	0	0	3	1	0	6	14	DISCHARGE	Y	N
62	1125632	35	2	0	0	3	0	0	5	14	DAMA	N	Y
63	1132230	20	2	0	0	3	0	1	6	16	DISCHARGE	Y	N
64	1146512	30	1	0	0	4	3	2	10	2	DEATH	N	Y
65	1142584	30	2	0	0	3	0	4	9	7	DISCHARGE	Y	N
66	1108226	35	3	2	0	3	3	1	12	48	DISCHARGE	N	Y
67	1089518	35	3	2	0	3	6	0	14	27	DAMA	N	Y
68	1100518	20	2	2	1	3	2	1	11	13	DISCHARGE	Y	N
69	811219	30	2	2	0	4	4	3	15	16	DAMA	N	Y
70	1097530	20	3	3	1	4	3	1	15	5	DEATH	N	Y
71	1078473	40	1	2	0	0	0	2	5	13	DISCHARGE	Y	N
72	1088794	30	3	0	0	2	0	0	5	6	DISCHARGE	Y	N
73	1132967	35	1	1	0	3	0	2	7	21	DAMA	Y	N
74	709606	20	3	0	4	4	3	2	16	8	DEATH	N	Y
75	102328	20	3	2	1	4	3	3	16	33	DAMA	N	Y
76	1016955	20	3	0	0	4	4	2	13	5	DEATH	N	Y
77	1088286	20	2	0	2	4	4	4	16	10	DEATH	N	Y
78	1090724	40	2	0	0	4	4	3	13	5	DEATH	N	Y
79	232443	20	2	0	0	4	4	4	14	14	DEATH	N	Y
80	1071146	35	2	0	0	3	3	2	10	9	DISCHARGE	Y	N
81	560295	30	1	0	0	2	0	4	7	6	DISCHARGE	Y	N
82	1022005	20	1	2	0	3	4	0	10	10	DAMA	Y	N
83	1005243	55	2	1	0	4	4	0	11	21	DISCHARGE	N	Y
84	975534	28	1	1	0	4	1	3	10	21	DISCHARGE	N	Y
85	1020128	20	1	0	0	2	0	1	4	7	DISCHARGE	Y	N
86	1018513	50	2	0	0	3	0	1	6	14	DISCHARGE	Y	N
87	1019045	20	2	0	0	4	4	0	10	8	DAMA	N	Y
88	984239	20	2	0	0	4	3	0	9	11	DISCHARGE	Y	N
89	1023853	20	2	3	2	4	3	0	14	26	DISCHARGE	Y	N
90	576311	45	2	0	0	3	2	1	8	12	DISCHARGE	Y	N
91	1081594	25	2	2	0	3	0	0	7	8	DISCHARGE	Y	N
92	1084339	20	2	1	0	4	1	0	8	19	DEATH	N	Y
93	1087151	30	1	0	0	3	2	3	9	13	DISCHARGE	Y	N
94	1067817	40	1	2	0	4	2	3	12	14	DISCHARGE	Y	N
95	669637	30	3	1	0	0	0	1	5	23	DISCHARGE	Y	N
96	1024042	30	2	1	0	4	4	4	15	18	DAMA	N	Y

S. No	MRD	INITIALS	AGE	AGE GROUP	SEX	DOA	DOD	FEVER	COUGH/ SPUTUM	PAIN ABDOMEN	DECREASED URINE OUTPUT	BURNING/ INCEASED FREQUENCY OF URINATION	LOOSE STOOLS	ALTRED SENSORIUM/ SIEZURES	SOB	VOMITING/ NAUSEA	OTHERS	DM	HTN	COPD/B.ASTHMA/ANY RESP DISORDER	CLD
97	135150	KB	65	5	M	07-08-2021	20-08-2021	N	N	N	N	Y	N	N	Y	N	CHEST PAIN	Y	Y	N	N
98	1134612	BS	56	4	M	09-07-2021	17-07-2021	Y	Y	N	N	N	N	N	Y	N	N	Y	N	N	N
99	1110017	BS	52	4	M	11-07-2021	18-07-2021	Y	Y	N	N	N	N	Y	N	N	N	N	N	N	N
100	653940	YC	57	4	M	04-04-2021	05-05-2021	Y	N	N	Y	N	N	Y	N	N	NON HEALING WOUND	Y	Y	N	N
101	1119576	ST	45	3	F	07-06-2021	14-07-2021	Y	N	N	N	N	N	N	Y	N	N	N	N	N	N
102	675667	S	77	6	F	02-04-2021	04-04-2021	N	N	N	Y	Y	N	Y	Y	N	N	Y	N	N	N
103	1051576	VS	68	5	M	01-04-2021	06-04-2021	Y	Y	N	Y	N	N	N	N	N	N	Y	Y	N	N
104	1114972	KK	24	1	F	30-05-2021	02-06-2021	N	N	N	Y	N	N	N	Y	N	PEDAL SWELLING	N	Y	N	N
105	414537	PA	76	6	M	20-05-2021	06-06-2021	Y	N	N	N	N	N	N	Y	N	N	N	N	N	N
106	1118160	PC	75	6	M	04-06-2021	22-06-2021	Y	N	N	Y	N	N	Y	N	N	N	Y	Y	N	N
107	1072186	PL	73	6	M	01-06-2021	09-06-2021	N	N	N	N	N	N	Y	N	N	LETHARGY	Y	Y	N	N
108	995225	GC	74	6	M	23-05-2021	08-06-2021	N	N	N	Y	N	N	N	N	Y	N	Y	Y	Y	N
109	1110017	BS	52	4	M	17-05-2021	08-06-2021	Y	N	N	N	N	N	Y	Y	N	LETHARGY	N	N	N	N
110	1143359	VK	45	3	M	27-7-2021	22-08-2021	N	N	Y	N	N	N	Y	N	N	N	Y	Y	N	N
111	1142389	VKJ	77	6	M	24-07-2021	31-07-2021	Y	Y	N	N	N	N	N	N	N	BLOOD IN SPUTUM	Y	Y	Y	N
112	1089471	NB	49	3	M	18-04-2021	07-06-2021	N	Y	N	N	N	N	N	N	N	CHEST PAIN	N	Y	N	N
113	654942	SK	75	6	F	23-10-2021	05-11-2021	Y	N	N	N	N	N	Y	N	N	N	Y	Y	Y	Y
114	1027500	PD	65	5	F	21-02-2022	27-02-2022	Y	N	Y	N	N	N	N	Y	N	N	Y	Y	N	N
115	1032043	JS	65	5	M	21-02-2022	01-03-2022	N	Y	N	N	N	N	N	Y	N	N	N	Y	N	N
116	1030532	GK	78	6	F	18-02-2022	28-02-2022	N	N	N	N	N	N	Y	N	N	RIGHT HEMIPARESIS	N	N	N	N
117	1141594	U	65	5	F	23-07-2021	05-08-2021	Y	N	N	N	N	N	N	N	N	LETHARGY	N	N	N	N
118	1131585	DD	58	4	F	03-07-2021	11-07-2021	N	N	N	N	N	N	N	Y	N	CHEST PAIN	Y	Y	N	N
119	363711	RA	73	6	M	24-02-2021	09-03-2021	N	N	N	Y	N	N	Y	N	N	LOSS OF APPETITE	Y	Y	N	Y
120	1038687	MK	20	1	F	03-03-2021	07-03-2021	Y	N	N	N	N	N	N	N	Y	N	N	N	N	N
121	1044183	HRC	81	6	M	08-03-2021	29-03-2021	N	N	N	N	N	N	Y	N	N	RIGHT HEMIPARESIS	Y	Y	N	N
122	1042210	HD	100	6	M	05-03-2021	18-03-2021	N	N	N	N	N	N	Y	N	N	DOMESTIC FALL	N	N	N	N
123	1007889	RU	70	5	F	03-01-2022	24-01-2022	N	N	N	Y	N	N	Y	Y	N	N	N	Y	N	N
124	244513	NK	68	5	M	18-01-2022	20-01-2022	N	N	N	Y	N	N	N	Y	N	N	Y	N	Y	Y
125	679538	JS	42	3	M	20-07-2021	29-07-2021	N	N	N	N	N	N	N	Y	N	HEADACHE	N	N	Y	N
126	1079301	HA	68	5	M	07-04-2021	15-04-2021	Y	N	N	N	N	N	N	Y	N	N	Y	Y	N	N
127	1105118	LS	23	1	M	08-05-2021	11-05-2021	Y	Y	N	N	N	N	Y	N	N	N	N	N	N	N
128	1074194	MS	58	4	M	03-04-2021	15-04-2021	Y	N	N	N	N	N	N	N	N	LEG SWELLING	Y	N	N	N
129	1013820	GS	37	2	M	18-01-2022	21-01-2022	Y	N	N	N	N	N	N	Y	N	N	N	N	N	N
130	1013169	SK	62	5	M	15-01-2022	28-01-2022	Y	Y	N	N	N	N	N	Y	N	N	N	N	N	N

S. No	MRD	CKD	CAD	HYPOTHYROIDISM	CVA	OTHER MEDICAL HISTORY	SURGICAL HISTORY	ALCHOL	SMOKING	OTHER SUBSTANCE	GCS	gcs GROUP	SBP	DBP	HR	RR	SPO2%	FIO2 %	RTI	UTI	GIT	SKIN	BLOOD STREAM	CNS-INFECTION
97	135150	N	N	Y	Y	N	N	N	N	N	15	3	100	70	98	32	92	100	N	Y	N	N	N	N
98	1134612	N	N	N	N	SIEZURE DISORDER	N	N	N	N	15	3	120	70	99	30	84	50	Y	N	N	N	N	N
99	1110017	N	N	N	N	PLHIV	N	N	N	N	3	1	60	30	89	20	62	21	N	N	N	N	Y	N
100	653940	Y	N	N	N	N	BELOW KNEE AMPUTATION	N	N	N	12	2	90	60	86	20	94	28	N	Y	N	Y	N	N
101	1119576	N	N	N	N	N	N	N	N	N	15	3	110	70	127	32	94	100	Y	N	N	N	N	N
102	675667	Y	Y	N	N	N	N	N	N	N	10	2	60	30	100	28	82	28	N	Y	N	N	N	N
103	1051576	Y	Y	N	N	N	POST CABG	N	N	N	14	3	110	70	116	28	95	100	Y	N	N	N	N	N
104	1114972	N	N	Y	N	N	N	N	N	N	14	3	90	60	117	28	84	21	N	N	N	N	N	N
105	414537	N	N	N	N	N	N	N	Y	N	15	3	110	70	98	26	92	50	Y	N	N	N	N	N
106	1118160	N	N	N	N	N	POST TURP	Y	N	N	11	2	90	60	117	26	94	21	Y	Y	N	N	N	N
107	1072186	N	Y	N	N	N	POST CABG	N	N	N	13	3	110	70	96	19	96	21	Y	N	N	N	N	N
108	995225	N	N	N	N	N	D. CRANIOTOMY, POST TRACHEOSTOMY	N	N	N	7	1	60	40	124	24	94	60	Y	Y	N	Y	Y	N
109	1110017	N	N	N	N	PLHIV	N	N	N	N	8	1	90	60	120	24	85	21	N	Y	Y	N	Y	N
110	1143359	N	N	N	Y	N	N	N	N	Y	5	1	110	70	78	18	100	40	Y	Y	N	N	Y	N
111	1142389	N	Y	N	N	N	POST CABG	N	N	N	15	3	120	70	104	26	92	50	Y	N	N	N	N	N
112	1089471	N	N	N	N	N	N	Y	Y	N	15	3	130	70	127	32	89	37	Y	Y	N	N	Y	N
113	654942	Y	Y	Y	N	N	N	N	N	N	7	1	100	60	74	18	100	40	Y	Y	N	N	N	N
114	1027500	N	N	N	N	HYPERTHYROIDISM	N	N	N	N	15	3	80	60	124	26	90	50	N	N	Y	N	N	N
115	1032043	N	N	N	N	N	N	Y	N	N	15	3	120	70	117	22	96	100	Y	N	N	N	N	N
116	1030532	N	N	N	N	N	N	N	N	N	7	1	90	60	74	16	99	100	Y	N	N	N	N	N
117	1141594	N	N	N	N	N	N	N	N	N	15	3	130	70	86	16	98	21	N	N	N	N	N	Y
118	1131585	N	N	Y	N	N	N	N	N	N	15	3	100	60	112	18	90	50	Y	N	N	N	N	N
119	363711	Y	N	N	Y	N	N	N	N	N	12	2	90	60	113	18	100	36	Y	Y	N	N	N	N
120	1038687	N	N	N	N	N	N	N	N	N	13	3	90	60	140	46	81	50	Y	N	N	N	N	N
121	1044183	Y	N	N	Y	N	N	N	N	N	12	2	100	70	97	28	93	36	Y	Y	N	N	Y	N
122	1042210	N	N	N	N	N	N	N	Y	N	3	1	100	70	88	16	81	21	Y	Y	N	N	N	N
123	1007889	N	N	-	N	RA	N	N	N	N	11	2	100	60	107	26	92	50	Y	Y	N	N	N	N
124	244513	N	Y	N	N	BED RIDDEN	POST PTCA	Y	Y	N	13	3	90	60	98	22	96	80	N	Y	N	N	N	N
125	679538	N	N	N	N	KYPHOSCOLIOSIS	N	N	N	N	14	3	120	70	114	14	92	36	Y	N	N	N	N	Y
126	1079301	N	Y	Y	N	N	POST CABG	N	N	N	15	3	100	60	107	28	94	50	Y	N	N	N	N	N
127	1105118	N	N	N	N	N	N	Y	Y	OPIOID	4	1	80	40	140	18	65	21	Y	N	N	N	N	Y
128	1074194	N	N	N	N	N	N	Y	N	N	15	3	90	60	107	32	94	36	N	N	N	Y	N	N
129	1013820	N	N	N	N	DEPRESSIVE ILLNESS	N	N	N	N	15	3	140	70	110	26	94	36	Y	N	N	N	N	N
130	1013169	N	N	N	N	PROSTATOMEGALY	POST TURBT	N	N	N	15	3	60	40	117	26	96	100	N	Y	N	N	Y	N

S. No	MRD	GENITAL INFECTION	TLC (X 1000/ UL)	HB (gm/dl)	PLATELETS (X 1000/ UL)	UREA (mg/dl)	CREATININE (mg/dl)	SERUM SODIUM (mmol/L)	S. POTASSIUM (mmol/L)	S. CHLORIDE (mmol/L)	S. CALCIUM	S. MAGNESIUM	S. PHOSPHORUS	TOTAL BILIRUBIN (mg/dl)	SGOT (U/L)	SGPT (U/L)
97	135150	N	5	8.3	173	66	5.76	140	5.34	106	8.6	2.95	5.78	0.62	18	8
98	1134612	N	17.3	13.7	224	29	0.68	135	4.56	99	8.2	2.44	3.36	0.6	79	79
99	1110017	N	9	9.1	110	26	1.37	134	4.52	103	8.8	2.74	8.6	1.13	99	81
100	653940	N	8.7	9.3	106	187	4.22	145	4.02	102	7.8	2.8	4.9	0.28	168	20
101	1119576	N	13.8	12	126	29	0.4	138	3.4	102	7.5	2.21	2.88	1.02	35	33
102	675667	N	9.4	9	478	108	3.64	128	5.94	91	8.6	2.14	7.76	2.5	2028	182
103	1051576	N	20	7.3	232	281	5.8	130	5.05	100	8.2	2.48	5.1	0.48	19	18
104	1114972	Y	14	10.4	150	52	1.21	137	4.08	103	-	-	-	0.37	36	14
105	414537	N	16	11.2	543	29	0.83	139	4.18	103	8.1	2.89	3.7	1.37	36	31
106	1118160	N	15.4	10.6	140	55	1.99	144	2.82	104	7.6	1.5	2.67	1.12	53	38
107	1072186	N	13.6	12.2	160	115	2.53	152	4.94	114	10.5	3.28	6.93	0.42	26	19
108	995225	N	32	8.5	234	61	1.77	127	4.7	97	6.6	1	2	0.94	319	171
109	1110017	N	6.4	5.9	90	50	0.78	145	4.43	111	8	4.24	3.35	3.3	289	496
110	1143359	N	18	11.6	80	18	0.7	137	2.84	99	8.1	2.27	2.5	0.64	83	94
111	1142389	N	11.2	11.4	169	49	1.11	127	6.26	98	8.8	1.79	3	1.55	19	24
112	1089471	N	25.9	10.2	205	42	0.63	137	5.19	99	8.3	-	-	0.54	33	89
113	654942	N	22.9	12.2	378	93	3.5	136	4.74	100	7.6	2.25	6.9	0.38	59	45
114	1027500	N	9.2	11.5	227	41	1.37	138	4.23	108	6.9	1.54	3.2	1.48	124	119
115	1032043	N	9.9	17.6	80	48	2.29	138	3.84	100	8.6	2.72	4	0.69	87	54
116	1030532	N	11.2	7.7	308	28	0.77	134	4.6	97	8.7	2.16	3.27	0.57	41	22
117	1141594	N	12.4	10.2	601	50	1.38	108	5.16	74	9.6	1.73	3.65	0.23	21	11
118	1131585	N	12.1	8.7	196	108	4.08	138	5.2	101	-	-	-	0.35	15	14
119	363711	N	10	13.1	130	209	1.97	174	4.75	136	8.7	2.73	5.35	0.7	99	133
120	1038687	N	17	10.9	154	92	5.56	138	3.48	107	8.7	-	-	3.71	192	262
121	1044183	N	9.5	8.5	120	200	4.58	137	4.03	110	6.9	2.01	7.72	1.5	120	49
122	1042210	N	10.2	8.3	155	87	1.3	148	4.26	116	7.9	2.04	2.02	1.14	93	29
123	1007889	N	13.9	7.3	134	151	3.76	139	3.66	105	7.6	2.3	4.37	1.35	201	27
124	244513	N	25	11.2	481	156	3.96	127	5.87	90	9.5	3.06	4.5	1.1	46	24
125	679538	N	5.7	16.6	120	29	0.68	139	4.87	92	8.7	1.93	3.55	0.83	26	30
126	1079301	N	26.7	11.1	250	79	3.29	138	6.4	104	-	-	-	1.04	39	63
127	1105118	N	13.5	14.8	194	186	3.93	125	3.76	91	7.2	3.09	7.1	0.86	38	26
128	1074194	N	10.3	11.9	212	155	1.41	138	4.52	107	7.9	2.8	5.8	1.24	60	53
129	1013820	N	12.7	13.8	202	25	2.97	139	3.61	100	-	-	-	0.89	24	40
130	1013169	N	11.1	11.7	40	102	1.29	155	3.69	115	8.3	1.7	2.57	0.66	16	22

S. No	MRD	ALP (U/L)	TOTAL PROTIENS (gm/dl)	S ALBUMIN (gm/dl)	ESR	CRP (mg/L)	PCT	HBV/ HCV/ HIV STATUS	B-1,3 GLUCAN ASSAY	GALACTOMANAN	ANTI-SARS COV2 ANTIBODIES	DENGUE/LEPTO/SCRUB	PROTEINS	GLUCOSE	RBC'S	PUS CELL
97	135150	123	6.8	3.69	-	-	-	-	-	-	-	-	3+	NEG	70-72/HPF	10
98	1134612	120	7	3.57	22	48.55	0.05	-	-	0.09	2.44	-	1+	NEG	NEG	NEG
99	1110017	93	7.7	3.26	57	-	10.83	-	523	0.48	-	-	1+	NEG	NEG	6
100	653940	329	5.2	2.37	-	-	1.5	-	102.37	-	0.02	-	3+	2+	3-4/HPF	NEG
101	1119576	82	4.5	2.85	-	33.95	0.07	-	143	-	45.28	-	2+	NEG	40-45/HPF	NEG
102	675667	226	9.2	2.85	120	108	31.38	NR	-	-	-	-	2+	NEG	5-7/HPF	2
103	1051576	72	5	2.16	-	311	5.1	NR	57	0.27	14.41	-	1+	NEG	90-100/HPF	NEG
104	1114972	158	6.1	3.07	40	1.89	0.05	HCV REACTIVE	-	-	-	-	3+	NEG	8-10/HPF	NEG
105	414537	78	5.8	2.92	68	147	0.16	-	179	0.39	-	-	1+	NEG	1-3/HPF	NEG
106	1118160	107	5.9	2.92	31	28	100	-	-	0.47	2.1	-	1+	NEG	35	100
107	1072186	87	7.8	4.17	73	2.76	0.08	NR	353	0.21	44.77	-	1+	NEG	70-80/HPF	6
108	995225	289	5.5	2.31	49	298	100	NR	-	-	-	-	TRACES	NEG	8-10/HPF	25
109	1110017	124	5.9	3.52	-	77	1.63	HIV REACTIVE	-	-	-	-	2+	NEG	15-20/HPF	6
110	1143359	311	6.3	2.47	10	-	0.42	NR	-	-	0.13	-	1+	NEG	2-3	NEG
111	1142389	111	6.6	3.21	70	173	0.28	NR	77.59	0.21	0.2	-	TRACES	3+	8-10	6
112	1089471	89	4.9	2.59	-	18	0.17	-	-	0.84	50.59	-	1+	NEG	3-4	NEG
113	654942	137	5.7	2.82	55	-	13.05	-	523	1.85	-	-	1+	1+	10-12	16
114	1027500	627	5.2	2.64	-	-	0.95	NR	-	-	-	-	1+	NEG	2-5	8
115	1032043	88	7.1	3.87	9	-	20.5	-	-	-	0.072	-	1+	NEG	40-50	4
116	1030532	128	6.8	3.9	19	-	0.07	-	-	-	-	-	NEG	NEG	150-180	4
117	1141594	166	8.3	3.81	120	41	-	-	-	-	-	-	TRACES	NEG	NEG	NEG
118	1131585	95	6.1	2.66	100	45	0.37	NR	-	1.52	25.27	-	3+	NEG	NEG	NEG
119	363711	133	7.4	3.02	53	-	1.91	-	-	-	151.4	-	2+	NEG	10-12	60
120	1038687	145	6.2	3.62	31	46	2.49	NR	76.78	0.92	0.12	-	2+	1+	10-12	NEG
121	1044183	211	5.7	2.31	-	62	0.51	-	79	0.78	-	-	1+	NEG	10-15	8
122	1042210	68	5.1	2.99	19	-	0.49	NR	107	0.19	-	-	TRACES	NEG	2-3	2
123	1007889	264	4.9	2.35	-	-	24.88	HCV REACTIVE	277	0.37	-	-	NEG	NEG	12-15	NEG
124	244513	119	6.7	2.57	81	-	3.28	-	-	-	-	-	3+	1+	60-65	25
125	679538	74	7	3.82	5	3.45	0.08	NR	-	-	-	-	NEG	NEG	NEG	NEG
126	1079301	213	6.3	3.02	78	314	24.35	NR	304	0.39	0.03	-	TRACES	3+	NEG	NEG
127	1105118	56	5.8	2.87	6	8.62	0.62	HCV REACTIVE	-	-	39.29	-	TRACES	TRACES	20-25	NEG
128	1074194	145	4.7	1.99	102	-	6.44	NR	-	-	-	-	TRACES	1+	30-40	NEG
129	1013820	77	6.5	3.28	62	91	0.13	-	-	-	-	-	-	-	-	-
130	1013169	105	5.5	2.72	47	64	6.85	NR	172	0.59	0.587	-	TRACES	NEG	5-8	25

S. No	MRD	CULTURE GROWTH_URINE	BLOOD	CSF	SPUTUM/ET SECREATION	PUS/WOUND SWAB	PLEURAL FLUID	ASCITIC FLUID	2D ECHO LVEF	EF	GHK/RWMA	LVID (S)	LVID (D)
97	135150	NEG	NEG	-	-	-	-	-	28	2	RWMA	47	54
98	1134612	-	--	-	KLEBSIELLA, ASPERGILLUS	-	-	-	36	2	GHK	42	49
99	1110017	NEG	NEG	NEG	-	-	-	-	22	2	GHK	48	58
100	653940	MORGANELLA	-	-	NEG	ACINETOBACTER	-	-	22	2	GHK	46	56
101	1119576	NEG	NEG	-	ACINETOBACTER	-	-	-	35	2	GHK	29	48
102	675667	NEG	NEG	-	-	-	-	-	20	2	GHK	35	45
103	1051576	-	-	-	-	-	KLEBSIELLA	-	40	2	RWMA	35	45
104	1114972	-	NEG	-	-	-	-	-	16	1	GHK	49	52
105	414537	-	-	-	ACINETOBACTER	-	-	-	32	2	GHK	50	60
106	1118160	NEG	NEG	-	ACINETOBACTER	-	-	-	28	2	GHK	41	49
107	1072186	NEG	-	-	KLEBSIELLA	-	-	-	32	2	RWMA	22	38
108	995225	NEG	E.COLI	-	ACINETOBACTER,KLEBSIELLA, PROTEUS	-	-	-	26	2	GHK	48	58
109	1110017	CANDIDA	KLEBSIELLA	NEG	NEG	-	-	-	35	2	GHK	38	48
110	1143359	PSEUDOMONAS	E.COLI	-	ACINETOBACTER	-	-	-	35	2	GHK	45	59
111	1142389	NEG	-	-	NEG	-	-	-	42	3	RWMA	35	49
112	1089471	PSEUDOMONAS	PSEUDOMONAS, STAPH EPIDERMIDIS	-	-	-	-	-	40	2	GHK	34	52
113	654942	NEG	NEG	-	ACINETOBACTER, CANDIDA	-	-	-	35	2	GHK	37	52
114	1027500	NEG	NEG	-	-	-	-	-	48	3	GHK	34	52
115	1032043	NEG	NEG	-	-	-	-	-	40	2	GHK	30	50
116	1030532	-	NEG	-	NEG	-	-	-	38	2	GHK	38	54
117	1141594	-	-	-	-	-	-	-	42	3	RWMA	34	49
118	1131585	NEG	NEG	-	-	-	-	-	28	2	RWMA	45	55
119	363711	PROTEUS	NEG	-	KLEBSIELLA	-	-	-	38	2	GHK	33	49
120	1038687	NEG	NEG	-	NEG	-	-	-	45	3	GHK	28	41
121	1044183	CANDIDA	CITROBACTER	-	CANDIDA	CITROBACTER	-	-	32	2	GHK	42	51
122	1042210	CANDIDA	-	-	ACINETOBACTER	-	-	-	42	3	GHK	34	49
123	1007889	CANDIDA	NEG	-	ACINETOBACTER, KLEBSIELLA	-	-	-	22	2	RWMA	50	59
124	244513	-	-	-	-	-	-	-	26	2	GHK	50	65
125	679538	NEG	-	-	-	-	-	-	42	3	GHK	34	48
126	1079301	NEG	NEG	-	-	-	-	-	28	2	RWMA	55	64
127	1105118	-	NEG	-	NEG	-	-	-	18	1	GHK	39	49
128	1074194	-	NEG	-	-	E.COLI	-	-	35	2	GHK	45	55
129	1013820	-	-	-	-	-	-	-	35	2	GHK	46	60
130	1013169	-	KLEBSIELLA, ENTEROBACTER	-	-	-	-	-	16	1	GHK	33	39

S. No	MRD	PA PRESSURE (PASP)	PAO2/FIAO2	PLATELETS	TOTAL BILIRUBIN	MAP	GCS	S. CREATININE/ URINE OUTPUT	SOFA SCORE ON INCLUSION DATE	TOTAL HOSPITAL STAY DURATION (DAYS)	OUTCOME	STATUS OF PATIENT AT 30 DAYS AFTER DISCHARGE ALIVE	DEAD
97	135150	20	2	0	0	4	0	4	10	14	DISCHARGE	N	Y
98	1134612	30	3	0	0	0	0	0	3	9	DISCHARGE	Y	N
99	1110017	35	2	2	0	4	4	2	14	8	DEATH	N	Y
100	653940	30	2	2	0	4	3	4	15	31	DAMA	N	Y
101	1119576	20	3	1	0	0	0	0	4	38	DAMA	N	Y
102	675667	20	3	0	2	4	4	4	17	3	DEATH	N	Y
103	1051576	35	3	2	0	4	4	3	16	6	DEATH	N	Y
104	1114972	20	3	1	0	3	2	1	10	4	DAMA	N	Y
105	414537	40	3	0	0	3	0	0	6	18	DISCHARGE	Y	N
106	1118160	30	1	2	0	4	3	2	12	19	DAMA	N	Y
107	1072186	28	1	2	0	3	1	0	7	9	DISCHARGE	Y	N
108	995225	30	3	1	0	4	2	0	10	17	DAMA	Y	N
109	1110017	20	1	2	2	4	3	1	13	23	DISCHARGE	Y	N
110	1143359	40	1	1	0	4	4	0	10	27	DISCHARGE	Y	N
111	1142389	30	1	0	1	1	0	0	3	8	DISCHARGE	Y	N
112	1089471	20	2	1	0	2	1	0	6	51	DISCHARGE	Y	N
113	654942	20	1	0	0	3	3	3	10	14	DISCHARGE	Y	N
114	1027500	50	2	0	2	4	1	2	11	7	DAMA	N	Y
115	1032043	20	1	1	0	3	0	2	7	12	DAMA	Y	N
116	1030532	30	1	0	0	4	4	0	9	11	DAMA	N	Y
117	1141594	50	0	0	0	1	0	1	2	14	DISCHARGE	Y	N
118	1131585	60	2	0	0	3	0	3	8	9	DISCHARGE	Y	N
119	363711	20	1	2	0	4	3	3	13	14	DAMA	N	Y
120	1038687	35	3	1	2	4	3	4	17	5	DAMA	N	Y
121	1044183	20	2	2	1	4	3	3	15	22	DAMA	N	Y
122	1042210	35	1	0	0	3	4	1	9	14	DISCHARGE	N	Y
123	1007889	36	2	2	1	4	4	2	15	21	DAMA	N	Y
124	244513	30	2	0	0	4	4	3	13	3	DAMA	N	Y
125	679538	20	1	2	0	1	1	0	5	10	DISCHARGE	Y	N
126	1079301	20	1	0	0	1	0	2	4	9	DISCHARGE	Y	N
127	1105118	20	2	0	0	4	4	3	13	4	DAMA	N	Y
128	1074194	25	1	0	1	4	0	1	7	13	DISCHARGE	Y	N
129	1013820	20	1	0	0	0	0	2	3	4	DISCHARGE	Y	N
130	1013169	45	2	4	1	4	1	0	12	14	DAMA	Y	N

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The Principal,
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Subject: **Approval of Plan of Thesis.**

This is to inform you that the Faculty of Medical Sciences in its meeting held on 29.10.2021 has approved the plan of thesis of **Hardeep Singh**, a student of your college for **MD General Medicine** along with the names of Supervisor/Co-Supervisor as under:-

Topic Plan of Thesis
To study the clinical profile and outcome of patients with sepsis and left ventricular systolic dysfunction.

Supervisor
Dr. Vipin Kumar
Asst. Prof.,
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Co-Supervisor

Dr. Dinesh Jain
Prof.,
Dept. of Medicine,
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Dr. Bhupinder Singh
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DMCH, Ludhiana.

The student has to submit the four copies of thesis (along with PDF format) , to the Controller of Examinations, BFUHS, Faridkot, at least six months before the commencement of theory examination, if he/she is otherwise eligible to take the examination. He /She must directed to attach a photocopy of this letter with the forwarding letter while submitting thesis to the University along with the letter of approval of University for change of supervisor/Co-Supervisor, change of topic,(if any).

S Singh

Dr. S P Singh MS(Oph)
Controller of Examinations

Note :

49. *If any discrepancy found regarding topic of thesis or Supervisor/Co-Supervisor, inform undersigned within one month after issue of this letter.*
50. *The student may submit the thesis as per the schedule already conveyed i.e for May/June examination 30th Nov of the preceding the year of examination and for Nov/Dec examinations 31st May of the year of examination.*

TO STUDY THE CLINICAL PROFILE AND OUTCOME OF PATIENTS WITH SEPSIS AND LEFT VENTRICULAR SYSTOLIC DYSFUNCTION

THESIS PROPOSAL

FOR APPROVAL OF SUBJECT OF THESIS TO BE SUBMITTED

IN PARTIAL FULFILMENT OF THE REQUIREMENTS

FOR THE DEGREE OF

MD

GENERAL MEDICINE

OF THE

BABA FARID UNIVERSITY OF HEALTH SCIENCES

FARIDKOT

MARCH 2021

DR HARDEEP SINGH

DEPARTMENT OF GENERAL MEDICINE

DAYANAND MEDICAL COLLEGE AND HOSPITAL

LUDHIANA

ABSTRACT OF PLAN OF THESIS

Title	TO STUDY THE CLINICAL PROFILE AND OUTCOME OF PATIENTS WITH SEPSIS AND LEFT VENTRICULAR SYSTOLIC DYSFUNCTION
For the degree of	MEDICINE
Name of the candidate	Dr Hardeep Singh
Supervisor	Dr Vipin Kumar, Assistant Professor, Department of Medicine, DMC&H Ludhiana
Co-supervisor	Dr Dinesh Jain, Professor, Department of Medicine, DMC&H Ludhiana Dr Bhupinder Singh, Associate Professor, Department of Cardiology, DMC&H, Ludhiana
Institution	Dayanand Medical College & Hospital, Ludhiana
University	Baba Farid University of Health Sciences, Faridkot

Introduction: Sepsis is defined as life threatening organ dysfunction caused by a dysregulated host response to an infection. Sepsis is an important cause of morbidity/mortality in in-hospital admitted patients. Presence of myocardial systolic dysfunction with decreased ejection fraction may exacerbate the already present hypotension, hypoxaemia and end-organ damage. This study will observe outcome of patients with sepsis and left ventricular systolic dysfunction.

Aim of Study: 1) To study the clinical profile of patients with sepsis and left ventricular systolic dysfunction. 2) To study the outcome of patients with sepsis and left ventricular systolic dysfunction.

Materials & methods: A prospective, single center, observational study to be carried out in Medical ICU/HDU at Dayanand Medical College and Hospital, Ludhiana of patients with Sepsis (Definition & criteria as per Third International Consensus Definitions For Sepsis and Septic shock,2016 –Sepsis 3 guidelines) and Left Ventricular Systolic Dysfunction. Duration of the study will be 1 year from March 1, 2021 to March 1, 2022. Sepsis patients with Left ventricular ejection fraction (LVEF) of less than 50% as an indicator of Left ventricular systolic dysfunction will be included in the study. All patients will be included in the study after complying with inclusion and exclusion criteria and written informed consent for admission into study has been taken from the patient or legal representative of the patient.

Clinical Significance: Sepsis and Left ventricular systolic dysfunction have their own associated morbidity/mortality outcomes in admitted patients. Combination of sepsis and systolic dysfunction may worsen the clinical outcome. Studying the effect of severity of LV systolic dysfunction in sepsis patients can help in better management of the these patients.

Keywords: Sepsis, Septic Shock, Left Ventricular Systolic Dysfunction, Left Ventricular Ejection Fraction (LVEF%).

Hardeep Singh

(Signature) Candidate | (Signature) Co-supervisor | (Signature) Co-supervisor | (Signature) Supervisor | (Signature) Principal

CERTIFICATE OF FACILITIES AVAILABLE

This is to certify that the facilities for work on the subject of thesis titled **"TO STUDY THE CLINICAL PROFILE AND OUTCOME OF PATIENTS WITH SEPSIS AND LEFT VENTRICULAR SYSTOLIC DYSFUNCTION"** exist in the Department of General Surgery, DMC&H, Ludhiana and will be provided to be candidate. We will see that the data being included in the thesis are genuine and is collected by the candidate himself under our supervision and guidance.

Supervisor

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Dr. Dinesh Gupta
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Date: 25/3/21

APPROVAL PROFORMA
BY RESEARCH & ETHICAL COMMITTEE
DAYANAND MEDICAL COLLEGE & HOSPITAL, LUDHIANA

Name of candidate	Dr. Hardeep Singh
Department	Medicine
Topic of Thesis	TO STUDY THE CLINICAL PROFILE AND OUTCOME OF PATIENTS WITH SEPSIS AND LEFT VENTRICULAR SYSTOLIC DYSFUNCTION
Likely date of appearing in PG examination	MAY 2023
Date of enrollment	JUNE 30, 2020
Name of Head of Department	Dr. Dinesh Gupta
1) **Supervisor** 2) **Co-Supervisor**	Dr. Vipin Kumar Dr. Dinesh Jain Dr. Bhupinder Singh
Signature of Members of Research Committee with Stamp	**Signature of Members of Ethics Committee with Stamp**
Dr. Harpreet Kaur (Professor & Head, Pathology)	Mr. R K Sharma (Legal Expert)
Dr. Jaspal Singh (Professor & Head, Surgery)	Dr. Ramit Mahajan (Associate Professor, Gastroenterology)
Dr. Dinesh Gupta (Professor & Head, Medicine)	Dr. Anu Sharma (Professor, Anatomy)
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Dr. Gurpreet S Wander (Vice Principal)	Dr. Gurpreet S Wander (Vice Principal & Professor & Head, Cardiology)
	Dr. Gagandeep Singh (Secretary, Ethics Committee Professor & Head, Neurology)

Approved: Yes / No

Dr. Sandeep Puri
Chairperson
Research Committee

Approved: Yes / ~~No~~

Prof. Arvind Malhotra
Chairperson
Ethics Committee

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PROFORMA FOR REGISTRATION OF SUBJECT FOR DISSERTATION

1	NAME OF THE CANDIDATE AND ADDRESS	Dr. Hardeep Singh Post Graduate Student Department Of Medicine Dayanand Medical College And Hospital, Ludhiana
2	NAME OF THE INSTITUTE	Dayanand Medical College and Hospital, Ludhiana
3	COURSE OF STUDY AND SUBJECT	Doctor of Medicine in General Medicine
4	DATE OF ADMISSION OF COURSE	30-June-2020
5	TITLE OF THE TOPIC	Clinical Profile And Outcome Of Patients With Sepsis and Left Ventricular Systolic Dysfunction.

6. BRIEF RESUME OF THE INTENDED WORK

6.1 NEED FOR THE STUDY

Sepsis is defined as life threatening organ dysfuncytion caused by a dysregulated host response to infection. organ dysfunction can be represented by an increase in sequential (sepsis-related) organ failure assessment (sofa) score of equal to or more than 2 points.[1]

Sepsis can occur by wide variety of micro-organisms. In a study done by Paary et.al. on patients of severe sepsis admitted in ICU-most common source of infection was from respiratory tract (37.2%), followed by urinary tract (10.3%), and intra-abdominal (9.5%).[2] The reported mortality of sepsis ranges from 30-40% and mortality of severe sepsis is over 50%.[3-7] In an Epidemiological study of sepsis patients in ICU, done by Chatterjee S et.al.- most frequent site of infection among severe sepsis patients admitted in icu was respiratory tract(53.3%), followed by abdominal (14.9%), blood stream (14.3%) and urinary tract infection (12.9%). Majority of infections were caused my gram negative microbes (73%), gram positive (12.6%), fungal etiology (6.2%), tropical etiology (7.2%).[8]

Presence of sepsis can lead to production of pro-inflammatory mediators (TNF-a, Interleukins IL-1,IL-6,cytokines etc)which can lead to vasodilation, endothelial cell contraction/damage, increased vascular permeability. Resulting decreased peripheral vascular resistance, intra-vascular fluid leakage, tissue edema can produce shock like conditions, all leading to decreased tissue perfusion. Endothelial cell damage and exposure of basement membrane collagen, connective tissue can lead to production of micro-thrombi. Sepsis can produce widespread micro-thrombi which exacerbate already present tissue hypo-perfusion.

IV fluids (along with anti-microbial drugs) are started in management of sepsis as soon as possible to maintain tissue perfusion. but if bp remain low even after receiving iv fluids, use of vasopressor drugs is indicated. These drugs constricts blood vessels and corrects the decreased peripheral vascular resistance caused by sepsis and helps increase blood pressure and maintain organ perfusion.

Septic shock can be clinically identified by a vasopressor requirement to maintain a mean arterial pressure of 65mmhg or greater and serum lactate level greater than 2mmol/l (>18mg/dl) despite adequate fluid replacement.(1) Use of multi-vasopressor drugs support may be needed to maintain organ perfusion. Otherwise decreased tissue perfusion of septic shock can produce multi-organ dysfunction like acute lung injury/ acute respiratory distress syndrome, acute kidney injury, septic encephalopathy, acute hepatitis which may warrant need for mechanical ventilator support, hemo-dialysis support etc.

Sepsis/septic shock can lead to spectrum of myocardial dysfunction which is caused by maldistribution of coronary blood flow, cytokine (TNF-alpha, IL-1BETA, IL-6) induced neutrophil activation and myocardial injury, complement (C5a) triggered myocyte contractility failure, calcium handling dysregulation and cytopathic hypoxia due to mitochondrial dysfunction. [9-11]

In a study done by pulido et.al. out of total 106 patients of severe sepsis/ septic shock , 64% patients developed myocardial dysfunction. 37% patients had LV diastolic dysfunction, 27% patients had LV systolic dysfunction, 31% had RV dysfunction, 4% had overlap of type of dysfunction, 10% had biventricular systolic dysfunction. [9]

Myocardial dysfunction pre-existing or sepsis induced can lead to decreased cardiac output and can increase hypotension, hypoxaemia, hypoperfusion of end organs .Evaluation of lvef as an indicator of cardiac systolic dysfunction using 2d-Echocardiography early can lead to better management of sepsis. Iv fluids given to maintain bp and perfusion of end-organs in sepsis are optimized depending on the severity of cardiac dysfunction as it can cause fluid overload.

6.2 REVIEW OF LITERATURE

According to The Third International Consensus Definition for sepsis and septic shock (sepsis-3), sepsis is defined as life threatening organ dysfuncytion caused by a dysregulated host response to infection. For clinical operationalisation, organ dysfunction can be represented by an increase in sequential (sepsis-related) organ failure assessment (sofa) score of equal to or more than 2 points. Sepsis is associated with an in-hospital mortality greater than 10%.[1,13]

Septic shock is defined as a subset of sepsis in which profound circulatory, cellular and metabolic abnormalities are associated with a greater risk of mortality than with sepsis alone. Patients with septic shock can be clinically identified by a vasopressor requirement to maintain a mean arterial pressure of 65mmhg or greater and serum lactate level greater than 2mmol/l (>18mg/dl) in the absence of hypovolemia(or adequate volume resuscitation) .Septic shock is associated with an in-hospital mortality rate of 40%.[1,13]

Sequential (sepsis-related) organ failure assessment score (sofa)

System	Score				
	0	1	2	3	4
Respiration					
PaO_2/FIO_2, mmHg (kPa)	⩾400 (53.3)	<400 (53.3)	<300 (40)	<200 (26.7) with respiratory support	<100 (13.3) with respiratory support
Coagulation					
Platelets, $\times 10^3$ μL^{-1}	⩾150	<150	<100	<50	<20
Liver					
Bilirubin, mg dL^{-1} (μmol L^{-1})	<1.2 (20)	1.2–1.9 (20–32)	2.0–5.9 (33–101)	6.0–11.9 (102–204)	>12.0 (204)
Cardiovascular	MAP ⩾ 70 mmHg	MAP < 70 mmHg	Dopamine < 5 or dobutamine (any dose)[a]	Dopamine 5.1–15 or epinephrine ⩽ 0.1 or norepinephrine ⩽ 0.1[a]	Dopamine > 15 or epinephrine > 0.1 or norepinephrine > 0.1[a]
Central Nervous System (CNS)					
Glasgow Coma Scale score[b]	15	13–14	10–12	6–9	<6
Renal					
Creatinine, mg dL^{-1} (μmol L^{-1})	<1.2 (110)	1.2–1.9 (110–170)	2.0–3.4 (171–299)	3.5–4.9 (300–440)	>5.0 (440)
Urine output, mL per day				<500	<200

FIO_2: fraction of inspired oxygen; MAP: mean arterial pressure; PaO_2: partial pressure of oxygen.
[a]Catecholamine doses are given as $\mu g kg^{-1} min^{-1}$ for at least 1 h.
[b]Glasgow Coma Scale scores range from 3 to 15; higher score indicates better neurological function.

In a study done by M. Mukhyaprana Prabhu et.al. source of infection not known in 42.8% patients. Among the known source, the most common source of infection was Pneumonia (24.2%).24.2% patients reported LV systolic dysfunction on the first day of admission. Out of 66 patients of septic shock ,mortality rate was 48.48% and among them survivors had better lvef than non survivors signifying that the low ef is a measure of mortality in these patients.11.76% of septic shock survivors had lvef <_50% and 43.75% of septic-shock non-survivors had lvef<_50%. Non-survivors exhibited lower mean blood pressure, lower ejection fraction. Among the inoptropes used for maintaining blood pressure, 44.1% of the survivors were resuscitation with Norepi and combination of noradrenaline and dopamine (59.4%) being the most often used in non-surviving septic shock patients. [14]

In a study done by Sua kim, Jung-Dong Lee et. al total of 208 patients were enrolled in the study. Pneumonia was the leading cause of Septic Shock (60% patients), followed by urinary tract infection (13.5% patients). In-Hospital mortality was 49.5% in total. APACHE-2 score of the non-survivors was higher than that of the survivors, the CK-MB and Troponin-T were higher in the non-survivors than in survivors. Risk of in-hospital mortality of patients with severe LV systolic dysfunction with LVEF <30% was significantly higher than that of patients with normal LV systolic function, although the risk of in-hospital moprtality did not change in patients with mild or moderate systolic dysfunction. [15]

In a study done by Gilbert Abou Dagher et. al. out of total 174 sepsis patients, 87 had previous history of congestive heart failure (CHF). 43.7% cass had respiratory infection, 33.3% had urinary tract infection, 11.5% had skin infection, 4.6% had GI tract infection. Patients with CHF were more likely to be admitted to ICU (59.8% vs 48.8%) as compared to control group. Patients with CHF had higher in-hospital mortality (57.5% vs 34.5%) and a higher 72 hour mortality (10.3% vs 4.6%).[16]

Definition of sepsis-induced cardiomyopathy (SIC) is a global, reversible, systolic and diastolic dysfunction of the left ventricle (LV) or right ventricle (RV), which is induced by myocardial depressants

released from pathogens and the host, and global ischemia after peripheral vasodilation, and arterial and capillary shunting in septic distributive shock. [17]

In a retrospective study done by Dong Geum Shin et.al. 366 sepsis patients were included. Sepsis patients were classified into 3 groups according to LVEF% in Echocardiographic assessment done within 48 hours of diagnosis of sepsis. LVEF <50% (n=36), LVEF 50-70% (n=252), LVEF >70% (n=78). Decreased LVEF occurred in 36(10%) patients and hyperdynamic LVEF >70% ocurred in 78 (21%) patients. Off 366 patients, 103 (28%) patients died. Mortality rates were similar in three groups, however mortality rates were significantly higher in patients with abnormal EF (decreased or increased vs normal). Length of hospitalisation was significantly shorter in patients with hyperdynamic EF. BNP and initial Tropoins were highest in patients with reduced EF. [18]

6.3 AIMS AND OBJECTIVES OF THE STUDY

1) To study the clinical profile of patients with Sepsis and LV Systolic Dysfunction (LVEF <50%).
2) To study the outcome of patients with Sepsis and LV Systolic Dysfunction (LVEF<50%).

7.0 MATERIALS AND METHODS

A prospective, single center, observational study to be carried out at Dayanand Medical College and Hospital, Ludhiana of patients with Sepsis (Definition & criteria as per Third International Consensus Definitions For Sepsis and Septic Shock,2016 –Sepsis 3) with Left Ventricular Systolic Dysfunction.Duration of the study will be from March 1 ,2021 to March 1, 2022 .Only patients admitted in Medical ICU/HDU will be included in the study. Sepsis patients will be screened for myocardial dysfunction using 2d-Echocardiography if clinically indicated. Left ventricular systolic dysfunction will be evaluated using 2D-Echocardiography and left ventricular ejection fraction LVEF<50% will be taken as criteria for systolic dysfunction.These Sepsis patients will be categorised into 3 groups- patients with LVEF<30%, LVEF30-40% and LVEF41-50%. All patients will be included in the study after complying with inclusion and exclusion criteria and written informed consent for admission into study has been taken from the patient or legal representative of the patient.

7.1 SOURCE OF DATA

Patients' demographic data, present and past history, clinical/ laboratory data echocardiography report, treatment and hospital course will be observed prospectively and will be noted on a proforma and observed/analysed for the study.

7.2 METHOD OF COLLECTION OF DATA

- **Inclusion Criterion:**

  -Age equal to or more than 18

  -Patients admitted in Medical ICU/HDU

  -Presence of infection caused by any bacteria/ virus/ fungus/ protozoa/ tropical fever microbial agents and evaluated using CBC, PCT, ELISA, serology, cultures, 1-3 beta D-Glucan, Galactomanan assay, smear microscopy,Imaging and other relevant laboratory tests.

  -Infection of any site- respiratory, urinary, abdominal, blood-stream, skin,etc.

-Criteria of diagnosing sepsis according to sepsis-3 guidelines 2016.

sepsis is defined as life threatening organ dysfuncytion caused by a dysregulated host response to infection. For clinical operationalisation, organ dysfunction can be represented by an increase in sequential (sepsis-related) organ failure assessment (sofa) score of equal to or more than 2 points

-Sepsis patients will be screened for LV Systolic dysfunction if clinicaly indicated (patients with high risk factors like-T2DM, HTN, Obesity ,etc/ ecg changes/ increased cardiac enzymes/ increased BNP/ clinical features of heart failure/ previous h/o heart failure or as per clinical judgement of the primary physician)and with LVEF<50% on 2D-Echocardiography as a marker of LV systolic dysfunction.

-With written consent from patient/legally authorised representative to be a part of study

- **Exclusion Criterion :**

  -age <18 years

  -Pregnant patients

  -poor echo window

  -patients with LV Systolic Dysfunction>50% LVEF

  -patients with congenital heart disease

Duration of Study/ Number of Cases: Duration of study will be from March 1,2021 to March 1, 2022

Outcome : Primary outcome of the study to be observed in terms of:

- Discharge/Mortality outcome
- Total hospital stay
- 30-day mortality outcome

Statistical Analysis:

data will be described in terms of range, mean+standard deviation (+SD),median, frequencies(number of cases) and relative frequencies(percentages) as appropriate. All statistical calculations will

be done using (statistical package for the social science) SPSS 21 version (SPSS incc., Chicago, IL, USA) statistical program for Microsoft Windows.

7.3 DOES THE STUDY REQUIRE ANY INVESTIGATIONS OR INTERVENTIONS TO BE CONDUCTED ON PATIENTS OR OTHER HUMANS OR ANIMALS? IF SO, PLEASE DESCRIBE BRIEFLY.

No.

7.4 HAS ETHICAL CLEARANCE BEEN OBTAINED FROM YOUR INSTITUTION IN CASE OF 7.3?

Yes

LIST OF REFERENCES

(1) Singer M, Deutschman CS, Seymour CW. The Third International Consensus Definitions for Sepsis and Septic Shock (Sepsis-3). JAMA. 2016;315:801-10.

(2) Paary TT, Kalaiselvan MS, Renuka MK, Arunkumar AS. Clinical profile and outcome of patients with severe sepsis treated in an intensive care unit in India. Ceylon Med J. 2016;61:181-4.

(3) Angus DC, Linde-Zwirble WT, Lidicker J, Clermont G, Carcillo J, Pinsky MR. Epidemiology of severe sepsis in the United States: analysis of incidence, outcome, and associated costs of care. Crit Care Med. 2001;29:1303-10.

(4) Finfer S, Bellomo R, Lipman J, French C, Dobb G, Myburgh J. Adult-population incidence of severe sepsis in Australian and New Zealand intensive care units. Intensive Care Med. 2004;30:589-96.

(5) Brun-Buisson C, Meshaka P, Pinton P, Vallet B; EPISEPSIS Study Group. EPISEPSIS: a reappraisal of the epidemiology and outcome of severe sepsis in French intensive care units. Intensive Care Med. 2004;30:580-8.

(6) Todi S, Chatterjee S, Bhattacharyya M. Epidemiology of severe sepsis in India. Crit Care. 2007;11:P65

(7) Mohan A, Shrestha P, Guleria R, Pandey RM, Wig N. Development of a mortality prediction formula due to sepsis/severe sepsis in a medical intensive care unit. Lung India. 2015;32:313-9.

(8) Chatterjee S, Bhattacharya M, Todi SK. Epidemiology of Adult-population Sepsis in India: A Single Center 5 Year Experience. Indian J Crit Care Med. 2017;21:573-7.

(9) Pulido JN, Afessa B, Masaki M, Yuasa T, Gillespie S, Herasevich V, et al. Clinical spectrum, frequency, and significance of myocardial dysfunction in severe sepsis and septic shock. Mayo Clin Proc. 2012;87:620-8.

(10) Flierl M.A., Rittirsch D., Huber-Lang M.S., Sarma J.V., Ward P.A. Molecular events in the cardiomyopathy of sepsis. Mol Med. 2008;14:327–36.

(11) Hochstadt A, Meroz Y, Landesberg G. Myocardial dysfunction in severe sepsis and septic shock: more questions than answers? J Cardiothorac Vasc Anesth. 2011;25:526–35.

(13) Mehta Y, Kochhar G. Sepsis and Septic Shock. Journal of Cardiac Critical Care TSS. 2017;1:3-.

(14) Prabhu MM, Yalakala SK, Shetty R, Thakkar A, Sitapara T. Prognosis of left ventricular systolic dysfunction in septic shock patients. J Clin Diagn Res. 2015;9:OC05-8.

(15) Kim S, Lee JD, Kim BK, Kim YH, Kim JH. Association between Left Ventricular Systolic Dysfunction and Mortality in Patients with Septic Shock. J Korean Med Sci. 2020;35:e24.

(16) Abou Dagher G, Hajjar K, Khoury C, El Hajj N, Kanso M, Makki M, Mailhac A, Bou Chebl R. Outcomes of patients with systolic heart failure presenting with sepsis to the emergency department of a tertiary hospital: a retrospective chart review study from Lebanon. BMJ Open. 2018;8:e022185.

(17) Antonucci E, Fiaccadori E, Donadello K, Taccone FS, Franchi F, Scolletta S. Myocardial depression in sepsis: from pathogenesis to clinical manifestations and treatment. J Crit Care. 2014;29:500-11.

(18) Shin DG, Kang MK, Seo YB, et al. Factors associated with abnormal left ventricular ejection fraction (decreased or increased) in patients with sepsis in the intensive care unit. PLoS One. 2020;15:e0229563.

PROFORMA

SERIAL NO.

NAME-_____S/D/W/H OF_____

AGE_____SEX_____DATE OF ADMISSION_____

MRD NO. _____HOME ADDRESS_____

PHONE NO._____CONSULTANT_____

DISCHARGE/DAMA/DEATH (ON DATE) _____

1.PRESENTING COMPLAINTS(WITH DURATION)

- FEVER
- COUGH/ SPUTUM/CHEST PAIN
- PAIN ABDOMEN
- DECREASED URINE OUTPUT
- BURNING MICTURITION/ INCREASED FREQUENCY OF URINATION
- LOOSE STOOLS
- ALTERED MENTAL STATUS/ SIEZURES
- OTHER

2.PAST HISTORY OF ILLNESS (WITH DURATION)

- DIABETES MELLITUS
- HYPERTENSION
- COPD/B.ASTHMA/ANY RESP DISORDER
- CHRONIC LIVER DISEASE
- CHRONIC KIDNEY DISEASE
- PAST MEDICAL HISTORY
- PAST SURGICAL HISTORY
- PERSONAL HISTORY- ALCOHOL/SMOKING/ SUBSTANCE USE

3.PRESENTING VITALS

- Sensorium
- Resp. rate

BP (mmHg)

Spo2/Fio2

Heart rate

4.EXAMINATION

- CNS
- CVS
- RESP
- ABDOMEN

SIGNATURE OF CANDIDATE

SIGNATURE OF SUPERVISOR i

5.DIAGNOSIS

6. LAB INVESTIGATIONS

TESTS	DATE-	DATE-	DATE-
TLC (X 1000/UL)			
N/L/M/E/B (%)			
HB (gm/Dl)			
PLATELETS (X1000/UL)			
UREA (mg/dL)			
CREATININE (mg/dL)			
Na/K/Cl (mmol/L)			
Ca/Mg/P			
BILIRUBIN (T/D) (mg/Dl)			
SGOT/SGPT (U/L)			
ALP (U/L)			
S.PROTIEN (g/dL)			
S.ALBUMIN (g/dL)			
ESR			
CRP (mg/L)			
PCT			
OTHER TESTS (IF DONE)			
HBV/HCV/HIV			
B-1,3 GLUCAN ASSAY			
GALACTOMANAN			
DENGUE/LEPTO/SCRUB			
ANTI-SARS COV2 Ab			
URINE R/E			
PROTIENS			
GLUCOSE			
RBC			
PUS CELLS			

SIGNATURE OF CANDIDATE **SIGNATURE OF SUPERVISOR ii**

7.SMEAR MICROSCOPY/ CULTURE GROWTHS/ELISA/ SEROLOGICAL TESTS/ PCR

SAMPLE	RESULTS	CULTURE SENSTIVITY
URINE		
BLOOD		
CSF		
SPUTUM		
PUS/ASPIRATION FLUID		
PLEURAL FLUID		
ASCITIC FLUID		

8. 2D-ECHOCARDIOGRAPHY FINDINGS

DATE	RESULTS
LVEF %	
LVID- SYSTOLIC/DIASTOLIC MR/TR/ANY VALVULAR DISEASE PA PRESSURE OTHER SIGNIFICANT FINDING	

9.SOFA SCORE

SOFA SCORE PARAMETERS	BASELINE SOFA SCORE	SOFA SCORE ON INCLUSION DATE
PaO2/FIO2 (mmHg)		
PLATELETS (X 1000 PER microL)		
TOTAL BILIRUBIN mg/Dl		
MAP[(2XDBP)+SBP/3] mmHg		
GCS		
S.CREATININE (mg/DL)		
URINE OUTPUT (ml/day)		
	TOTAL SCORE=	TOTAL SCORE=

10. TOTAL HOSPITAL STAY DURATION (DAYS):

11. DISCHARGE/DAMA/DEATH (ON DATE):

12. STATUS OF PATIENT AT 30 DAYS :

SIGNATURE OF CANDIDATE **SIGNATURE OF SUPERVISOR** iii

INFORMED PATIENT CONSENT

Date: _____

I ..... D/o, W/o, S/o .....
..... am willing to participate in the study **"TO STUDY THE CLINICAL PROFILE AND OUTCOME OF PATIENTS WITH SEPSIS AND LEFT VENTRICULAR SYSTOLIC DYSFUNCTION"** The procedure with its complications has been explained to me in local vernacular language. I have fully understood that no special tests and extra expenditure in any form, merely for the purpose of thesis is being carried out. I am free to opt out the study any time without assigning any reason.

....
Signature of the Witness Signature of the patient

Name:

Signature of Candidate **Signature of Supervisor**

मरीज अनुमति फार्म

तिथि _____

मैं _____ पत्नी/पुत्र/पुत्री _____

निवासी _____

इस शोधकार्य "TO STUDY THE CLINICAL PROFILE AND OUTCOME OF PATIENTS WITH SEPSIS AND LEFT VENTRICULAR SYSTOLIC DYSFUNCTION" में भाग लेने के लिए सहमत हूँ। मैं इस शोधकार्य में भाग लेने के लिए अपनी स्वेच्छा से तैयार हूँ। मैं पुष्टि करता/करती हूँ की यह स्वीकृति देने के लिए मुझे कोई आर्थिक प्रलोभन नहीं दिया गया और न ही दबाव डाला गया है। मुझे इस शोधकार्य का उद्देश्य स्पष्ट कर दिया गया है और मुझे यह विश्वास है की यह विज्ञान और मानवता के लाभ के लिए है। मुझे इस शोधकार्य में होने वाले जोखिम के बारे में समझा दिया गया है और मेरी समझ में यह जोखिम बहुत मामुली है।

मैं इस शोधकार्य में भाग लेने के सम्बन्ध में किसी भी समय अपनी अस्वीकृति व्यक्त कर सकता/सकती हूँ और मेरे अस्वीकृति व्यक्त करने का मेरे इलाज पर कोई प्रभाव नहीं पड़ेगा।

_____ _____

गवाह के हस्ताक्षर रोगी के हस्ताक्षर/अंगूठे का निशान

नाम: नाम:

_____

अन्वेषक के हस्ताक्षर

_____

विद्यार्थी के हस्ताक्षर

ਮਰੀਜ਼ ਰਜ਼ਾਮੰਦੀ ਫਾਰਮ

ਮਿਤੀ_____

ਮੈ_____ਪਤਨੀ/ਪੁੱਤਰ/ਪੁੱਤਰੀ_____

ਵਾਸੀ _____

"TO STUDY THE CLINICAL PROFILE AND OUTCOME OF PATIENTS WITH SEPSIS AND LEFT VENTRICULAR SYSTOLIC DYSFUNCTION" ਤੇ ਚਲ ਰਹੀ ਖੋਜ ਵਿਚ ਹਿੱਸਾ ਲੈਣ ਲਈ ਰਜ਼ਾਮੰਦ ਹਾਂ। ਇਸ ਖੋਜ ਕੰਮ ਵਿਚ ਹਿੱਸਾ ਲੈਣ ਲਈ ਮੈ ਆਪਣੀ ਮਰਜ਼ੀ ਨਾਲ ਤਿਆਰ ਹਾਂ। ਮੈ ਇਹ ਦਸਣਾ ਚਾਹੁੰਦਾ ਹਾਂ/ ਚਾਹੁੰਦੀ ਹਾਂ ਕੇ ਇਹ ਰਜ਼ਾਮੰਦੀ ਦੇਣ ਵਾਸਤੇ ਮੈਨੂ ਕਿਸੇ ਵੀ ਤਰਾਂ ਦਾ ਲੋਭ-ਲਾਲਚ ਨਹੀ ਦਿੱਤਾ ਗਿਆ ਅਤੇ ਨਾ ਹੀ ਕੋਈ ਦਬਾ ਪਾਇਆ ਗਿਆ ਹੈ। ਮੈਨੂ ਇਸ ਖੋਜ ਦਾ ਮਨੋਰਥ ਸਮਝਾ ਦਿੱਤਾ ਗਿਆ ਹੈ ਅਤੇ ਮੈਨੂ ਇਹ ਭਰੋਸਾ ਦਵਾਇਆ ਗਿਆ ਹੈ ਕੇ ਇਹ ਖੋਜ ਵਿਗਿਆਨ ਅਤੇ ਮਾਨਵਤਾ ਦੀ ਭਲਾਈ ਲਈ ਹੈ। ਮੈਨੂ ਇਸ ਖੋਜ ਵਿਚ ਹੋਣ ਵਾਲੇ ਖਤਰੇ ਬਾਰੇ ਸਮਝਾ ਦਿੱਤਾ ਗਿਆ ਹੈ ਅਤੇ ਮੈਨੂ ਇਹ ਯਕੀਨ ਹੈ ਕੇ ਇਹ ਖਤਰਾ ਬਹੁਤ ਮਾਮੂਲੀ ਹੈ। ਮੈਨੂ ਇਸ ਖੋਜ ਕੰਮ ਵਿਚ ਹਿੱਸਾ ਲੈਣ ਦਾ ਢੰਗ – ਤਰੀਕਾ ਸਮਝਾ ਦਿੱਤਾ ਗਿਆ ਹੈ।

ਮੈ ਇਸ ਖੋਜ ਕੰਮ ਵਿਚ ਹਿੱਸਾ ਲੈਣ ਬਾਰੇ ਆਪਣੀ ਰਜ਼ਾਮੰਦੀ ਕਿਸੇ ਵੀ ਸਮੇ ਵਾਪਸ ਲੈ ਸਕਦਾ ਹਾਂ/ ਸਕਦੀ ਹਾਂ ਅਤੇ ਮੇਰੀ ਰਜ਼ਾਮੰਦੀ ਵਾਪਸ ਲੈਣ ਦਾ ਮੇਰੇ ਇਲਾਜ ਤੇ ਕੋਈ ਅਸਰ ਨਹੀ ਹੋਵੇਗਾ।

_____
ਗਵਾਹ ਦੇ ਹਸਤਾਖਰ

ਮਿਤੀ_____

ਪਤਾ _____

_____
ਮਰੀਜ਼ ਦੇ ਹਸਤਾਖਰ

ਮਿਤੀ_____

ਪਤਾ _____

_____
ਨਿਰੀਖਕ ਦੇ ਹਸਤਾਖਰ

_____
ਸਿਖਿਆਰਥੀ ਦੇ ਹਸਤਾਖਰ